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STRABISMUS

STRABISMUS

ITS ETIOLOGY AND TREATMENT

BY

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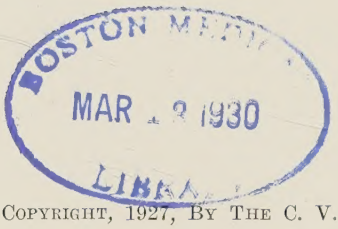

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AFFECTIONATELY DEDICATED

TO THE MEMORY OF

MY PARENTS

DUNCAN A. WILKINSON

AND

JANE FLOYD WILKINSON

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PREFACE

This book has been written in the interest of the cross-eyed child. The strabismic child is invariably neglected until he arrives at such an age that his condition is incurable.

The object of this book is to impress upon the public, the general practitioner and the oculist, the importance of early definite treatment of these cases, and to warn them of the evil that "watchful waiting" brings to this afflicted class.

I have presented the Etiology of Strabismus at length in order that the student of squint may obtain a broader view of the subject.

I have presented the chief operative procedures with discussions, endeavoring to give without bias their merits, difficulties, and defects, as seen in my practice. Finally, I have presented accurately measured operative procedures, with results to be expected.

I wish here to express my great appreciation for the invaluable assistance rendered me by a number of friends, in the way of research, proof-reading, typing, and criticisms, and particularly to the artist for the illustrations.

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STRABISMUS

CHAPTER I

HISTORICAL REMARKS ON STRABISMUS

So disfiguring a defect as strabismus did not escape the attention of the very earliest medical writers. Only a mere mention of the condition is to be found however, and no special treatment is recommended, as it was considered a permanent defect—an affliction or punishment sent by a displeased deity.

Hippocrates[1] noted the paralytic type in certain nervous conditions. He also observed the comitant variety, and considered that it was due to epilepsy, or that it was an hereditary taint, observing that a parent with a cross-eye often had a cross-eyed child.

The next step in the history of squint was made by Galen[2] who, in 131-210 A.D., gave a fairly good description of the ocular muscles, as follows:

"There are two (muscles) on the sides, at each canthus one, and two others, of which one is above, the other below. At a point where they run out into tendons, all four together form a complete circle, a broad tendon which terminates in the region of the ciliary body. Moreover, as it was well to have the eyes capable of rotating, nature added to the others (the recti) two obliques."

It was left for Fallopius in the sixteenth century to give us a more accurate description of the extra ocular muscles.

Paulus Aegineta, a noted Greek medical writer (seventh century), advised the use of a mask placed over the face, with small perforations over each eye. He had an idea that this might in some way induce the patient to look through these small apertures and thus straighten his eyes. Ambroise Paré, the most celebrated French surgeon of his day (1509 A.D.), took up Aegineta's idea and had masks made of horn with central perforations. (Fig. 1.)

Paré attributed squint to defective position of the child in the cradle, or to imitation of the nurse or companion; ideas which are still held by the laity.

Antoine Maître-Jan, 1707,[3] states that some authors claim that "malposition of the crystalline lens is the cause of squint, while others maintain as causes imaginary vices of the visual spirits. Some claimed that it is due to contraction or spasm of the ocular muscles," so we see our muscular theory began at least as early as 1707. This

same author discourses on refraction and mentions the fact that objects look larger to patients who squint. (Accommodation theory of Donders.)

Prior to the early part of the eighteenth century the treatment of squint was of no more avail than that recommended by Hippocrates, Galen, and Celsus. At this time, from 1727 to 1737, appeared the bombastic John Taylor, an itinerant oculist, of Norwich, England, who first introduced his operation in 1727. He traveled through Europe "curing" squint by cutting the conjunctiva at the inner canthus, occluding the good eye to cause the use of the squinting one. In the *Mercury* of France for June, 1737, there is the following announcement:

"Dr. Taylor, oculist to the King of Great Britain, has just arrived at Paris at the London Hotel, rue Dauphine, where he proposes remaining till the beginning of July, after which he will leave for

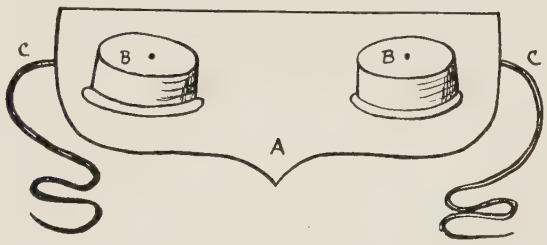

Fig. 1.—Strabismus mask, or spectacles, employed by Ambroise Paré. (From *The American Encyclopedia of Ophthalmology.*)

Spain. He requests us to publish the discoveries he has made, of straightening squinting eyes by slight and almost painless operation, and without fear of accident."

There is evidence that he did put eyes straight by operating, but what he really did will remain a mystery.

We take the following description of Taylor's operation as given by Lucien Boyer,[4] who quotes an address of LeCat's of Rouen:

"This refined and amiable man came to Rouen * * * and within a few days became the object of general admiration. He had an arsenal of superb instruments and handled them with great dexterity. He showed portfolios filled with authentic and highly commendatory credentials. The door to his hotel was guarded by soldiers and it was necessary for one to have an introduction in order to visit him. His operations were done in the midst of a brilliant circle of select persons.

"The great operation, the most marvelous of all, was that by which he proposed to straighten squinting eyes. His method was as follows: With a needle of silk he caught a portion of the conjunctiva of the squinting eye at the inferior part of the globe, and having made a loop of this silk, he used it to draw toward him that portion of the

conjunctiva which it included, which he cut with the scissors; then he applied a plaster to the sound eye; the squinting eye at once righted itself, and every one cried out, 'A miracle.'

"I availed myself of the freedom which he accorded me to inquire the motive for an operation which appeared to me to be absolutely useless, not to say dangerous. He replied that an eye only squinted because the equilibrium between its muscles was destroyed, and that to reestablish this equilibrium it was necessary only to weaken the muscle which dominated the others and that this is what he did in cutting one of the nerve filaments which was distributed to this powerful muscle."

Stevens[5] states:

"Whatever may have been Taylor's method, whether he divided the superior oblique as Heuerman asserts, or the inferior oblique as others state, whether the brilliant and clever man described by LeCat really only cut a fold of conjunctiva, it is certain that Taylor's work had no influence in introducing the surgical method of treatment to the world. If he divided one of the oblique muscles as he was supposed to do, he did not correct the squint, and if he simply divided the conjunctiva and some supposed nervous filaments which were assumed to supply a muscle, he still did not cure the squinting. There remain two possibilities: First, that the secret of the whole operation lay in the fact stated by LeCat; he covered the sound eye with plaster; the squinting eye became straight, and the people cried, 'A miracle.' Second, and it is much more probable, however, that he cut the internal rectus, but that he did not confide too much in his not-well-informed critics."

An event of historical importance in the treatment of squint was the recommendation of Buffon,[6] 1743, to cover the good eye and force the use of the squinting one, and thus improve its vision and aid in the cure of squint. He also recommended the use of glasses in certain conditions; thus he might be said to have been the first to inaugurate the two most beneficial and most frequently used nonsurgical aids in the treatment of squint; i.e., *padding the good eye and bringing up the acuity of vision in the bad one,* and *the use of glasses;* the latter so masterfully brought forward by Donders and perfected particularly by American ophthalmologists. His remarks are so significant and important that we shall quote them at length:

"One ordinarily attributes this effect (squint) to a defect of the correspondence between the muscles of each eye; the difference of the movement of its muscles, which, not acting in concord, produce the false direction of squint eyes. * * *

"M. de la Hire, and many others after him, have thought that squint is not caused by the defect of equilibrium or of correspondence between the muscles, but that it arises from a defect in the retina; they have claimed that the side of the retina which responds to the extremity of the optical axis was much more sensitive than all the rest of the retina; objects, they said, impressed themselves dis-

tinctly only on this most sensitive part, and, if this part is not found to correspond exactly with the extremity of the optical axis, in one or the other of the eyes, they turn aside and produce squint, by necessity where one will have to turn them in such a manner that their optical axes can get at this more sensitive and ill-placed part of the retina. But this opinion has been refuted by many physicians and in particular by Jurin. Indeed, it seems that de la Hire has not paid attention to what happens to squinting persons when they close the good eye, for then the squinting eye does not remain in the same situation, as it should if that situation was necessary for the extremity of the optical axis to get at the more sensitive part of the retina; on the contrary, this eye rights itself in order to aim directly at the object and try to see it; consequently, the eye does not turn aside to find this part claimed to be the most sensitive of the retina, and one must search for another cause for the defect. * * *

"But the most general cause, the most ordinary, of squint, and of which nobody that I know of has made mention, is the inequality of strength of the eyes. A small degree of inequality will cause an object seen by the stronger eye to be as distinctly perceived as if it had been seen by both eyes; a little more inequality will render the object when seen by both eyes, less distinct than if it were seen by the stronger eye alone; and finally, a great deal of inequality will render the object seen by both eyes so confused that in order to see distinctly one will be obliged to turn the weak eye, and put it in a position where it cannot hinder. * * *

"But, some will say, it is not positive that the inequality of the strength of the eyes must produce squint, for squinting people can be found whose eyes have equal strength; moreover, this inequality does in truth spread confusion of the objects, but this confusion need not make the weak eye turn aside, for, from whichever way it is turned, it always receives other images which would hinder it when one regards directly.

"Besides, I do not claim that the inequality of the strength of the eyes is the only cause of squint—I say further that it is a cause, the effect of which is necessary, so that it is perhaps not possible to cure a person of this defect whose eyes have too much unequal strength. * * *

"But when the eyes, although of unequal strength, have not, however, the degree of inequality which we have determined by the formula above, one can find a remedy for squint; it seems to me that the most simple, the most natural and perhaps the most efficacious of all the methods is to cover the good eye for a time; and the deformed eye would be obliged to act and to turn itself towards the objects, and take on this movement habitually in a short while.—But before making use of this on a patient, the degree of the inequality of the strength of the eyes must be ascertained, because it will never be successful except in eyes that are only a little unequal. * * *

"I do not then see how anyone can find a remedy for crossed eyes when they are crossed because of their too great inequality of strength —one would perhaps be able to cure by beginning with covering the good eye for some time, in order to give to the bad eye the direction and all the force which the defect of habit may have served to deprive it of, and then by prescribing glasses of which the glass before the bad eye

shall be plain, and before the good eye convex; insensibly, this eye would lose some of its strength and consequently would be less able to act independently of the other.

"It is evident from all that we have said above, that one cannot squint with both eyes at the same time—but there are some persons, who, without squinting both eyes at the same time, sometimes alternately squint first one eye and then the other—I do not believe that one can remedy this defect, except by wearing glasses, of which one of the lenses should be convex and the other concave, in proportion to the strength or weakness of each eye; but one would need to have more experience in this than I have, to be sure of any success * * *

"Before ending this Memoire, it is well to observe one thing essential to the judgment which one should bring to the degree of inequality of strength of the eyes of squinting persons; I have found in all my experience, that the squinting eye which is always the more feeble, gains strength by exercise, and that many persons whose squint I judged incurable because in the first tests I found too great a degree of inequality, having covered the good eye for a few minutes only, and having consequently been obliged to exercise the bad eye during this short time, they were themselves surprised at the amount of strength the bad eye gained, so that in measuring, after this exercise, the range of this eye, I have found it more extended, and have judged the squint curable; so in order to pronounce with some measure of certainty on the degree of inequality of the eyes and on the possibility of remedying the defect of squinting eyes, it is necessary first to cover the good eye for a time, in order to oblige the bad eye to exercise and gather up all its forces, after which one will be better able to judge the cases where one can hope that the simple remedy which I have proposed may succeed."

Erasmus Darwin[7] indorses the idea of Buffon that squint is due to defective vision in one eye. He says:

"One eye being less perfect than the other, on which account the patient endeavors to hide the worst eye in the shadow of the nose that his vision by the other may not be confused."

He also gives us the cover or pad treatment as follows:

"If the squinting has not been confirmed by long habit, and one eye be not much worse than the other, a piece of gauze stretched on a circle of whalebone, to cover the best eye in such a manner as to reduce the distinctness of vision of this eye to a similar degree of imperfection with the other, should be worn some hours every day, or the better eye should be totally darkened by a tin cup covered with black silk for some hours daily."

About this time Tenon,[8] in 1806, gave us that most comprehensive anatomy of the orbit, and in particular the description of the capsule which bears his name, and this information without doubt has had much to do with subsequent treatment of squint.

The first definite surgical effort in the treatment of squint was made in the first third of the nineteenth century; Delpech's operation of

dividing the tendon of Achilles in 1816 might be called the forerunner of tenotomy. In 1827 Anthony White suggested the cure of squint by means of a tenotomy. Pauli, of Landeau, was probably the first to attempt a tenotomy of the internal rectus on the living, as recommended by Anthony White. This was a failure. The operative procedure was more fully described by Stromeyer in 1836 in his treatise on *Subcutaneous Orthopedics,* but for want of courage or opportunity he never performed his operation on the living subject. The first authentic tenotomy for relief of squint was performed by Dieffenbach, of Berlin, on the twenty-sixth day of October, 1839. Dieffenbach performed on the living subject the operation that had been performed on the cadaver by Stromeyer.

The following graphic description of Dieffenbach's tenotomy of an ocular muscle for the relief of squint is given by Howe:[9]

"The head of the patient, a boy of seven, being fixed against the chest of one assistant, and the lids being held apart by hooks in the hands of a second assistant, thereupon I pushed a third hook through the conjunctiva, at the inner angle of the eye and quite through the deep cellular tissue there, and that hook I gave to a third assistant. Then I struck a fine double hook into the sclerotic at the inner angle of the eye, and holding it in the left hand, drew the globe as far outward as possible. I incised the conjunctiva, close to the globe where it is spread out at the inner angle of the eye. Going deeper, I dissected the cellular tissue from the ball, and then divided the muscle, with a pair of fine eye scissors, close to the globe."

Dieffenbach's first and second operations are described in the *Medizinische Zeitung,* November, 1839, as follows:

"Cutting through the conjunctiva and separating the connective tissue, then dividing the internal rectus muscle, using scissors, close to its insertion. The eye was immediately drawn outward by the external rectus, as if it had received an electric shock; and in another instant became straight.

"In his second case the operation was performed as in the last case, the conjunctiva being cut through and the sclerotic laid bare to the extent of four lines, in order to bring the muscle into view, which was cut with a curved scissors as before."

P. Bennett Lucas[10] described his method of operation as follows:

"After dividing the conjunctiva and the connective tissue to the desired extent, the blunt hook being inserted beneath the muscle, the operator transfers it to the left hand, and having brought the tendon into view, he divides it with a sharp pointed pair of scissors as close to its insertion as is compatible with the safety of the sclerotic."

Mr. Liston[11] gives his method of tenotomy as follows:

"With a little dissection the muscle is seen just as it ends in its tendon, and with a pair of scissors it is cut across close to its insertion into the sclerotic."

One observes that these procedures are not unlike the tenotomies as practiced in many of our American clinics at the present day. As Stevens suggests, however, it is hardly possible to have done a very close tenotomy with the instruments used at that time. Fig. 2 taken from Mackenzie,[11a] 1855, would indicate that the muscles were severed and not the tendon, as is practiced at the present day.

"The operator now takes the blunt hook in his left hand, and carrying the handle of it towards the temple, with the scissors he immediately divides, in ordinary cases, the tendon of the muscle from below upwards, and nearer the caruncula than where it is over the hook, as is represented in Fig. 2.

"In this manner of operating, the muscle will most frequently be divided just where the tendinous part meets the fleshy fibers.

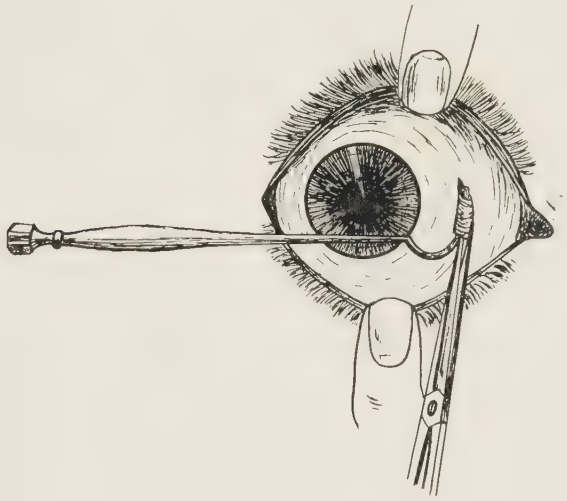

Fig. 2.—Mackenzie's method of tenotomy. (From Mackenzie, *On the Eye.*)

"If the distortion is slight, the handle may be carried over the nose, and the tendon divided nearer the cornea than where it lies over the hook, and close to its insertion."

Notwithstanding the above remarks and the accompanying cut, when we consider the dexterity of the operators and read their repeated statement, "Cut close to the sclerotic," we would infer that the first tenotomies were tenotomies proper, and not myotomies, in that they cut the muscle "close to the sclera." In their zeal to get more effect, they soon began to cut wider into the capsule and farther back on the muscle. Dieffenbach himself was one of the first to do a myotomy. He says:[12]

"If the conjunctiva be divided over a greater arc and toward the back of the globe, if the cellular tissue be extensively separated and the muscle be detached far back and divided at its middle, then the

eye, even in cases in which the whole cornea was hidden in the internal angle, stands quite straight after the operation."

This cutting of the belly of the muscle and the complete detachment of the muscle and capsule were attended by such glaring deformities, consisting of external deviation, sunken caruncle, and exophthalmos, that this operation for squint came into bad repute.

About this time A. von Graefe admonished his colleagues to adopt a more conservative procedure. He described his method of operating, which differed very little from the original method of Stromeyer. He advised opening the conjunctiva closer to the cornea, making a small opening, and doing a tenotomy as near the sclera as possible and using afterward a conjunctival stitch. This operation is extensively practiced today. (See Chapter XI.)

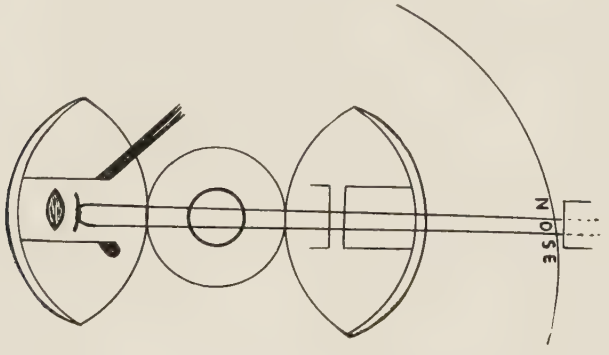

Fig. 3.—Von Graefe's Faden operation. (From Beard's *Ophthalmic Surgery.*)

These defects of tenotomy caused another operation to be developed —that is, a strengthening of the weaker muscle. This was first undertaken by Dieffenbach to relieve an external deviation produced by a myotomy done by himself in 1842; however, this was not a success. Guerin, of Nancy, in 1849, gave a more practical procedure. This he applied to a case of secondary squint.

The more accurate description of an advancement operation was by A. von Graefe in 1857. This was his famous "Faden operation." (Fig. 3.)

The same year G. Critchett, of London, described his three-stitch operation, of which Beard has said, "might be called the parent of most modern advancement operations." In 1897 Landolt made some improvements on Critchett's advancement, and his operation, with slight modifications, is extensively practiced at present. Almost every advancement operation since has been more or less a modification of Critchett's or Landolt's. (See Fig. 4.)

From this date up to the present time there have been devised many ingenious methods of shortening the weaker muscle and making it cope with the overacting one, and this has been done by muscular advancements (Critchett, Landolt, Weber, de Wecker, Prince, Verhoeff, Worth, et al.); capsular advancements (Knapp, de Wecker, Parinaud, Fox); resections (Vieusse, Schweigger, Prince, Reese); reefs (Savage, Valk, B. Harman, et al.); and by tucks (Todd, Bishop, Bruns, Clark, Briggs, et al.), but in one and all of these was the element of inexactness in the preoperative measurement of the squint, and want of proper gauging of the operation, and hence a variance of the end-results obtained. Then to obtain results there was the partial tenotomy and advancement—a combination of operations, both of which were inaccurate and left just as much to be guessed as heretofore.

It was this element of inexactness and want of definite measurements which prevented ophthalmologists from operating on children

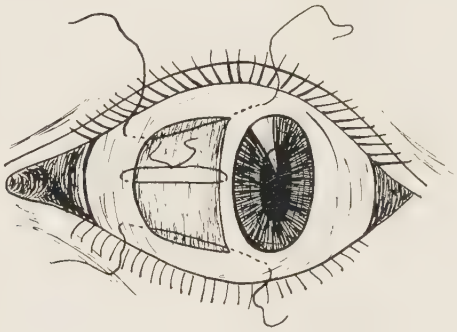

Fig. 4.—Critchett's method of advancement. Tissue excised and sutures introduced ready to tighten. (From *The American Encyclopedia of Ophthalmology.*)

at an early age before the vicious habits were formed and before it was too late to secure the aid of nature in procuring that all-important function, binocular vision, or fusion of the two images, to aid us in obtaining and maintaining straight eyes.

Parinaud[13] states that myotomy, introduced by Dieffenbach, 1839, constitutes the dominant fact in the history of squint. Of this he says:

"This historic importance is for two quite different reasons; myotomy, at the same time that it has inaugurated the truly efficacious treatment of squint, has completely falsified the ideas of its nature.

"Myotomy has falsified the ideas by giving a base, apparently solid, to the muscular theory of squint, which is the fundamental error on which we have existed until recent years. Without doubt, it has always been supposed that a muscular trouble was the cause of the deviation; but, since de la Hire and Buffon, there has been a tendency to explain squint as a functional trouble, not a mechanical one."

Thus we see that while the friends of Dieffenbach were following the natural tendency of trying to remove the supposed cause of squint by

cutting the supposedly offending muscle, there were other *theories,* some yet unborn, which were destined to take their part in the etiology and treatment of squint. De la Hire and Buffon saw some other cause than the overacting muscle or a weak external muscle. De la Hire saw a defect in the retina. May we not say the seed of the fusion theory, which was yet unknown, was here sown? And Buffon saw in 1743 the inequality of the two eyes and even recommended use of glasses, though admitting he did not yet know how to advise their use. Here we see the theory of hypermetropia as an etiologic factor and glasses as a means of cure which was later brought forward by Donders.

Böhm[14] observes that "Squinters can distinguish a certain print with the aid of convex glasses at a greater distance than with the unaided eye"—and came near the discovery of the relation between hypermetropia and squint.

Von Graefe[15] observed that there was a disturbance of accommodation, and this caused an "irregular contraction upon the affected muscle," and he also observed certain cases in which "pathologic convergence occurred only with accommodation for near objects." He recognized that the accommodation and convergence and the refraction all had some relation to certain cases of squint, but it was left for Donders to definitely explain these relations—which he did in his classic *Anomalies of Accommodation and Refraction,* 1864. He here establishes a definite relation between squint and hypermetropia and its connection with convergence. We quote from Donders as follows:[16]

"If we now inquire whether the cause of squint was ever before sought in hypermetropia, this question may be answered in the negative."

In this statement Donders definitely announces himself the author of this theory. His work can be said to be the second great step in the etiology and treatment of squint, and to this day his teachings have probably more followers than any other.

In 1873 Krenchel[17] first introduced the expression, "Innervation of convergence," and opposed this to the theory of muscular defect. After him Hansen Grut insisted upon the rôle of innervation of convergence, and combated the muscular theory. Mules and Berry sustained the same idea.

Von Graefe noted the insufficiency of the ocular muscles as a cause of squint, but did not stress the nervous relation to it. Javal made many observations and did much study in an effort to elucidate this theory, but it was left for Parinaud to more fully express the "error of the innervation of convergence" as a cause of squint, and his ideas are more clearly expressed by Duane, under the subject of "Excess of Convergence and Insufficiency of Divergence."

About the same time that Donders was so ably presenting his theory of the cause of squint and his treatment by the use of glasses, there developed another idea as to the etiology of squint; the so-called fusion theory of squint had its origin. De la Hire prepared the soil in the eighteenth century; Buffon sowed the seed; Javal, that indefatigable worker, and after him Parinaud, who first of all comprehended the entire nervous relation of squint, tilled the ground and brought forth the fruits of the fusion theory, which was so ably harvested and presented by C. Worth and is at present known as "Worth's fusion theory."

Of Javal's work, Parinaud said:[13]

"The stereoscope had just been invented by Wheatstone, in 1835, and Javal was the first one to apply it in the cure of squint. His idea was to teach the eyes to work together so that they would get their tendency back to associate their movements.

"He made a careful study of the condition of the amblyopic eye. He found that the amblyopia is not equally distributed on the retina; the peripheral parts are nearly normal, while the center or yellow spot is the real focus of the affection; therefore, we might say that an amblyopic eye sees only with its periphery; its acuity of vision after correction, 1/60 to 3/60, is in accordance with this. Therefore it is clear why the eye wanders around when the better eye is covered; it has lost its tendency to fixate because the function of the yellow spot is replaced by that whole periphery."

No one has done more than C. Worth[18] to perfect the fusion theory, and his views on this subject are ably presented in his *Manual on Squint*, 1903, and are backed by many careful observations made during many years of arduous labor.

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CHAPTER II

ETIOLOGY OF STRABISMUS

Probably no one subject in ophthalmology has been discussed more than the etiology of squint. No two ophthalmologists would today give even approximately similar definitions as to its cause. These two facts go to show the protean origin of squint and would suggest the varieties of treatment that may be necessary for the relief of different cases. Hence, it is not strange that so very many things have been mentioned as etiologic factors, and just as numerous have been the remedies for its relief.

Not until the beginning of the nineteenth century was there any worthy definite theory of squint promulgated, and since that time there have been *three theories* which have occupied the field of squint etiology, all of which still have ardent advocates, and all of which have elements of truth.

I shall present these three theories at some length, in order that the student of squint may get a broad view of its etiology, as sound and thorough understanding of etiology must of necessity precede rational treatment. After presenting these, I shall introduce a *fourth theory*, which I feel is justified, as it comprises the dominant idea of many, if not the majority, of the leading writers on squint. This theory I shall call the *Nervous Theory.*

The three outstanding theories as to the cause of squint which have dominated the treatment of that affection are the following:

1. **The Muscular Theory,** which attributes the defect to an overacting muscle. This theory held entire sway from the earliest age to the middle of the eighteenth century.

2. **The Accommodation Theory of Donders** claims that squint is produced by disturbance of the relation of convergence and accommodation due to hypermetropia or to hypermetropia and astigmatism. This theory, first suggested by Buffon in 1743, further elucidated by Böhm and von Graefe, made possible by the works of Helmholtz and Donders in optics, was finally firmly established by Donders about the middle of the last century.

3. **The Fusion Theory,** first suggested by Javal, further elucidated by Parinaud, more definitely stated by Verhoeff, and finally introduced and established by C. Worth, of London, is known in this country as Worth's Fusion Theory.

A fourth theory which I wish to introduce is the **Nervous Theory.** This was rather definitely stated by Mackenzie in 1845, and has been

more or less directly or indirectly admitted by every writer on squint during the past half century.

Many things have been mentioned as causal factors in squint. Every fond and apologetic parent can give a definite cause in his child's case, and want of concurrence in these opinions makes you an unworthy consultant. The chief of these are spasms, frights, falls, imitations of others, measles, mumps, whooping cough, nervousness, a spot on the nose, looking at a curl, injury at birth, and many others of less importance, some of which probably acted as contributing causes, while many others had no etiologic action. There are many conditions, such as congenital palsies, congenital defects of vision, central retinal diseases, congenital cataracts, scars on the cornea, spasms, injury at birth, which are, no doubt, contributing causes of squint. These will be discussed under individual headings.

We shall now discuss at some length the four theories above mentioned, taking them up in the order named.

The Muscular Theory of Squint

The advocates of this theory maintain that comitant squint is due to a too strong overacting rectus muscle, that the muscle is either too large and strong as compared with its antagonist, or that on account of its peculiar advantageous insertion, its shortness, or overdevelopment, or the shape of the orbit, it acts in excess of its antagonist and causes the eye to turn either *in* or *out*. This was the only outstanding theory of squint for many centuries and it still has many brilliant and ardent advocates. It must contain an element of truth to have lived so long and to be able to flourish at this enlightened age. We shall now review some of the opinions of its advocates.

In the early part of the eighteenth century Stromeyer published a book on orthopedic surgery and showed the beneficial effects of tenotomy in certain contracted tendons, and suggested that the severing of the tendon of the rectus muscle would relieve squint. In 1838, Dieffenbach, following this suggestion, did a tenotomy in a case of squint, and was successful in relieving the patient of his deformity. This was soon repeated on many patients by different men and often the operation was apparently a success. This at once had a tendency to firmly establish the muscular theory of squint. Of this Parinaud[1] says:

"However, after the work of Stromeyer (1838), Dieffenbach, Florient Cunier (1839-40), Jules Guerin (1841), Bonnet (of Lyons), Lucien Boyer (1841-43), the success of myotomy was proved and at the same time the muscular theory of squint is affirmed.

"Jules Guerin says clearly that the theories of squint ought to be subordinated to the results of the operation:

" 'If it is true, and experience does not allow me to doubt the fact, that section of the muscles of the eye rapidly and definitely cures squint, it follows that we should revise our theories of the deformity and make them accord with the results of the new method.'

"De Graefe is more radical and admits the muscular theory exclusively. According to him, 'This affection ought to be considered as disproportion between the average length of the muscles.' It matters little to the symptom that this inequality results from an abnormal insertion of the tendons or from modification of the structure of the muscles.

"In France, Giraud-Teulon brought forth the same arguments to exclude nervous influence. According to him, comitant squint is synonymous with mechanical squint. In the present day (1897) it seems, especially in the German school by Schweigger, Hauner, Schneller, Alf. de Graefe, etc., that muscular theory has found new defenders. It is manifest that on the old continent these defenders are less and less convinced, and that some, much bound to their past, work hard to conceal their conversion."

We shall now review the opinions of a few American authors. Reber[2] says:

"I believe with Dr. Posey, that there is much in anatomic peculiarities, whether of the build of the muscle, of the insert of the muscle, or of orbital build in children with alternating strabismus. I believe that the combined theory which will take account of Donders' theory, of Worth's fusion faculty, and of the anatomic possibilities, will account for practically all of the cases except congenital palsies."

Swan Burnett (Valk)[3] says of convergent squint:

"This condition is the legitimate result of the greater power and activity of those (interni) muscles."

Francis Valk,[3] who was probably the greatest muscular advocate in America, seems undecided at one moment and very positive in another. He says:

"I do not think that the true cause of strabismus has been fully explained, nor do I believe the 'last word has been said,' for if I ask what is the true or primal cause of a deviation of the optic axes, that is to say, what abnormal element is found in all cases, I find no answer in our textbooks today. Hence, in my opinion, hyperopia or fusion defects, per se, are contributing causes of squint, but they must have an imbalance of muscular power to rotate the eyes about their respective center, to cause squint, and that the innervation of the muscles is the simple child of a too active brain.

"I do not feel satisfied that we should say this case is due to an insufficiency of one function, or an excess of, or want of, innervation of the ocular muscles is the cause of this divergence, but I am more convinced that it must be in the anatomical construction and insertion of the ocular muscle, probably congenital."

Valk further says:

"I believe it was primarily due to the muscular condition of the eyes; in other words, the power of the straight muscles to move the

eyes in the field of vision and in the field of fusion. This has been called the Anatomical Muscular Theory and in my opinion is the true cause of all cases of heterophoria or heterotropia."

Savage's[4] explanation of esophoria as a cause of esotropia is so clearly stated by him that I shall quote it at length. He says:

"It is safe to say that without esophoria there can be no esotropia; [Savage considers esophoria of muscular origin] but ordinarily esophoria must be aided by one or more of the other causes in the production of esotropia. Esophoria, when high in degree, may eventuate in esotropia, and at the usual age or earlier, when there is no hyperopia, even when the refractive error is myopic. When esophoria alone is the cause of esotropia, the actual turning occurs when the externi give up the task of so antagonizing the interni as to make binocular vision possible. At this time the mind selects one eye for use, and the other is allowed to turn into the position predetermined by the excessive power of its internus. If its plane of action coincides with the horizontal plane of the eye, the position assumed will be directly in toward the nose; if the plane of its action is elevated, its attachment being too high, the eye will turn up as well as in and will be torted in; but if its plane of rotation is depressed, its attachment being too low, the eye will be turned down as well as in, and will be torted out. At the time of the beginning of the in-turning, the mind, in the interest of single vision, begins the work of suppressing the images on the retina of the in-turned eye. If the esotropia is not periodic or alternating, the work of mental suppression is soon accomplished, the patient becoming mind-blind in the crossed eye. When an esophoria alone is the cause of esotropia, the error probably shows itself earlier than usual. Spontaneous recovery, in a case, will never occur."

Savage[4] is of the opinion that comitant squint is always due to a previously existing heterophoria. Of this he says (p. 8083):

"When the weaker muscle of the pair no longer responds to the impulse from its fusion center, the stronger muscle causes the eye to turn into an abnormal or unharmonious position. Without the previous existence of heterophoria, there could be no comitant heterotropia."

Duane has the following to say with reference to the muscular theory:[5]

"A moderate proportion of cases of squint do, however, *develop out of a true muscular defect.* Thus, some cases of convergent squint, both in childhood and in adult life, develop out of a primitive paralysis of the abducens which has been converted into a comitant deviation by consecutive overaction and contracture of the internus (see page 742). Still more frequently it happens that a vertical deviation due to congenital insufficiency of the superior rectus (less often of other muscles) is the starting point of a convergent squint, or divergent strabismus (see pages 757 and 788). Furthermore, a convergent squint due to a convergence-excess may be aggravated by the presence of a primi-

tive weakness of the externi or overdevelopment of the interni. Again, a divergent squint may be produced by developmental changes in the orbit, causing a relative preponderance of the externi (see page 781), or by weakness of the internus due to operation. Such factors, however, play a subordinate part in the production of squint."

A. S. Green and L. D. Green[6] state:

"Ample evidence may be brought forth to show that, in the vast majority of cases, squint is a muscular or anatomic anomaly, not primarily a lack of faulty development of the fusion faculty."

A modified form of the muscular theory which deserves special mention is the *declination theory* of the late George T. Stevens.[7]

Dr. Stevens' theory, which he has made very attractive, was that squint was primarily due to a declination of the vertical axes of the eye, usually associated with hyperphoria. He considered that the disturbance due to declination upset the relation between convergence and accommodation and caused an excess stimulus to the internal muscle, causing the eyes to *turn in*. The declination is primarily due to a faulty insertion of one of the extraocular muscles. This theory is partially upheld by Jackson.

No one can doubt that marked declination of an image would materially interfere with its proper fusion and thus upset the normal relation between convergence, accommodation, and the general nervous control of the whole act of binocular vision. Whether this by itself could upset the equilibrium of the two eyes enough to cause squint may be questioned, but that it would act as an important aid to some other causative factor, as hyperopia, no one could doubt. A hyperphoria of high degree might cause at times such confusion of images as to cause the eye to turn in more markedly in the presence of other causative agencies.

Stevens[7] has the following to say with reference to the cause of strabismus:

"Converging strabismus is, in not a few instances, largely the result of the instinctive effort to force the visual line into the same horizontal plane in case of a normal tendency of one of these lines to rise above the other. This condition of hyperphoria or perhaps of hypertropia, in which the image of one eye tends to appear above the other or actually rises above, is a state of the vision for the relief of which the most strenuous efforts will be made.

"That this is in many cases true is shown by the fact that a tenotomy properly done on a superior rectus, if the upward rotation of that eye is excessive, will, in an important proportion of strabismic cases, at once and without further procedure relieve the convergent squint.

"In a much greater number of cases there is marked anotropia. The rotation upward of a very large percentage of cases of convergent squint exceeds 45 degrees and often exceeds 50 degrees of arc.

Here the extreme upward tendency of the eyes imposes upon the inferior recti an excessive tension in the effort to bring the visual lines to the ordinary planes of direction, and this tension is against the normal tension of the superior recti. Owing to the peculiar insertion of the superior and inferior recti muscles, this excessive tension constitutes a balance of force, in a majority of cases, against the external recti which is not easy to resist.

"But with excessive anophoria there are generally, or at least very often, extreme declinations, and to these extreme declinations, declinations which are materially emphasized and increased by the normal torsions in looking down with any degree of convergence, more than to any other one cause the strabismic deviations are due. If the eyes were not set too high, or if no hyperphoria existed, the patient might control the declinations, the state of anophoria would not induce a squint."

Stevens has reported a number of cases supporting his theory and has shown interesting and convincing photographs. The reader is referred to his excellent, painstaking work above cited.

Savage[4] does not concur in Stevens' theory as to declination and hyperphoria being the chief factors in determining squint. On page 8097, l.c., he has the following to say:

"The foundation for every permanent esotropia is intrinsic esophoria. An esotropia caused by hyperopia alone will be periodic, and never permanent. * * * The plane of action of the superior and inferior recti is such that they not only turn the eyes, respectively, up and down, but they also turn them in. Imbalance of the superior and inferior recti, whether that imbalance is shown in a hyperphoria of one eye and a cataphoria of the other, or in a double hyperphoria or in a double cataphoria, will become a factor in the production of esotropia only when there is an esophoria."

However, in his book on Ophthalmic Myology (page 421), Savage[8] does give cyclophoria or declination as a cause. He says:

"A sixth complication, if not a cause (of squint), is a plus or minus cyclophoria."

Opposition to the Muscle Theory.

Hansell and Reber[9] said:

"It may be well to repeat that squint is not primarily a defective muscular condition, and even less is it an affection that can be attributed to any one muscle. It is a matter of convergence power as related to divergence power, or, still more accurately, it is to be referred to as the muscular response of excessive or deficient nerve stimulation."

Parinaud[10] has the following to say:

"Hansen Grut has very correctly remarked that the straightening of the eyes under the action of chloroform is incompatible with the idea of shortening, an anatomical modification of the muscle. The

same remark applies, moreover, to these cases of hypermetropia which are corrected with glasses or with atropine. It is evident that in cases of this nature the trouble is purely nervous."

Parinaud[1] says:

"The primitive alteration of the muscles, their congenital feebleness, does not play any part at all in the pathology of squint. What one calls the insufficiency of the straight internals, is insufficiency of innervation of convergence. If the cause of that insufficiency is situated in the muscles, it ought to show itself with regard to the associated movements of direction, as well as in regard to those of convergence; now it is not so, as Desmarres has long since observed.

"It is, moreover, antiphysiological to admit that a muscle is, from birth, too feeble for the work which it should do normally. Of all the organs, the muscle is the one which adapts itself most easily to the need of its function. This adaptation is not only dynamic, but anatomic; it bears on its size, and in its length. The experiences of Marey, whose results have been recently confirmed by Joachimsthal, of Berlin, show that, if one displaces the insertions of a muscle, if one modifies the length of the lever to which it is inserted, the muscular fiber stretches or contracts permanently in order to adapt itself to the new conditions of functioning."

It is this very adaptability of the ocular muscle to new situations that permits us to operate so freely without bad results. That squint is not usually due to an overacting muscle is evident from the fact that the excursion of the eye is normal. In a recent case of alternating or periodic squint, there is *no* limitation of version in *any* direction, nor is there in constant comitant squint until the squint has been present for several years. The very fact that the muscles can and do move the eye into any desired position without limitation shows that there is no defect either in the muscles or their nerves. The further proof advanced by many is the fact, often observed, that the eye in most cases of squint at once becomes practically or quite straight when the patient is asleep or is placed under a general anesthetic. The very fact that the use of glasses and atropin will cure something over 30 per cent of cases also disproves the muscular theory. It must be further observed that the muscular system invariably adjusts itself to the demands made upon it: hence, if squint is due to a weak externus, the externus should develop to sufficient strength to prevent the in-turning of the eye. The demands made of the muscles of a blacksmith's arm invariably make his arm stronger; why does not the external rectus get stronger and hold the eye straight if it is a muscular defect? Demands made on a muscle make it stronger and not weaker. Let it be granted we have an overacting anatomically overlarge and strong internal rectus; why does it take this muscle three years (the average age of internal squint) to find out it is so strong? One would naturally expect it to dominate the turn-

ing of the eye even in the first year of life before the fusion faculty had an opportunity to develop and binocular vision is acquired.

In examining the pull of the interni by means of scales described elsewhere, in 250 cases of squint in children under ether anesthesia, I have never found any marked difference in the strength of the two interni. The internal rectus of the deviating eye gave practically always the same degree of resistance as was found in the straight one, and each had a normal pull, i.e., between 65 and 75 grams. This was true in all recent cases. In cases of long standing there is often a marked difference. This goes to prove that the overacting muscle is the *result* of faulty position of the eyes extending over many years, and not the *cause* of the deviation. It is my opinion that the anatomic peculiarities are seldom the primary cause of squint, but act only as contributing causes; and even this is very difficult to demonstrate in cases of *recent* squint.

The Accommodation Theory of Donders

The earliest suggestion of an optical defect as a causal factor in the production of squint was made by de la Hire as quoted by Buffon (Javal). Buffon in 1743 wrote a long dissertation in which he ascribes squint to an inequality in the strength of the two eyes. He even went so far as to suggest the use of glasses as a cure for squint; however, he modestly admitted he did not know how to determine the proper glass. Böhm, 1845, suggested the use of glasses to improve the vision in squint cases and came near making the discovery made by Donders, that many cases of squint can be relieved by the use of glasses.

Donders in the middle of the nineteenth century firmly established the fact that there was a close relation between convergent squint and excessive accommodation and convergence due to hypermetropia or hypermetropic astigmatism. He, at the same time, advocated the cure of squint by means of the correction of hypermetropia and astigmatism with glasses. Since his classical work on *Anomalies of Refraction and Accommodation,* published in 1864, this idea of squint has been known the world over as *Donders' Theory,* or the *Accommodation* or *Hypermetropic Theory of Donders.*

In order that we may understand Donders' idea of squint, I shall quote him at length:[11]

"We now know that by far the greater number of cases of strabismus are connected with anomalies of refraction (p. 292).

"1. Strabismus convergens almost always depends upon hypermetropia.

"2. Strabismus divergens is usually the result of myopia.

"Since in strabismus convergens hypermetropia in general exists, no other connection is conceivable than that hypermetropia is the cause

of the deviation. Hypermetropia is, indeed, the primary anomaly, to be sought in the structure of the eye, and originally proper to the organ; strabismus is the secondary condition which does not arise until some years after birth (p. 293).

"Hypermetropia is a very widely spread anomaly. I am convinced that it occurs still more frequently than myopia. Now, if strabismus convergens is in general the result of hypermetropia, the latter evidently is very often met with without strabismus; we may even say that only in a comparatively small number of cases of hypermetropia is strabismus developed. This, however, need not by any means surprise us. In general, in fact, the necessity of seeing an object *single* with both eyes together, is deeply felt. The direction of the visual lines is thereby forcibly determined. Of this I convinced myself years ago, in my experiments on the action of prismatic glasses. If we bring a weakly prismatic glass with the refracting edge turned inward before one of the eyes, the fixed object is directly seen double, but increased convergence is immediately involuntarily produced which makes the double image coalesce; and if, some moments later, we again remove the glass, double images for an instant reappear, which, however, equally rapidly disappear, in consequence of lessening the convergence. Now, it is as if the double images, of their own accord again coalesce; the movement takes place so spontaneously that the person is not even conscious of it. This abhorrence of double images, or rather, the instinctive adherence to binocular vision, preserves most hypermetropic individuals from strabismus. They sacrifice the advantages of seeing accurately, rather than to allow that on the yellow spots different objects should form their images. In this, therefore, we find the reason why not nearly all hypermetropics squint. If one eye be covered with the hand, while it, as well as the other, is open, the visual line will in *most* hypermetropics rapidly deviate inward. The same thing takes place when an ammetropic person holds a negative glass before the uncovered eye.

"The question which now suggests itself is: what circumstances must cooperate to give rise to strabismus in hypermetropic individuals? These circumstances are of a twofold nature: (a) those which diminish the value of the binocular vision; (b) those which render the convergence easier.

"To the first class belong:

"1. *Congenital difference in the accuracy of vision or in the refractive condition of the two eyes.* In hypermetropia the accuracy of vision is often imperfect, whether in one or both eyes. This is in part attributable to astigmatism, in part to a still unknown imperfection of the retina. If the diminished accuracy of vision affects only *one* eye, then, on too great convergence, the image of this eye will not so much disturb vision. The same is the case when the degree of hypermetropia in the deviating eye is greater, and the image in this eye is therefore less accurate. In either case, consequently, strabismus will more easily arise. But the tendency doubly increases when both circumstances, a higher degree of hypermetropia and diminished accuracy of vision, as is often the case, occur combined in the same eye. If the eye has long been deviated, there arises a secondary diminution of the accuracy of the vision as a result of strabismus, to which I shall sub-

sequently revert. In that case, however, we can, with the aid of the ophthalmoscope, often demonstrate a still higher degree of hypermetropia of this eye.

"2. *Spots on the cornea.* It is often remarked, that in oblique vision the deviated eye, or, indeed, both eyes, exhibit opacities or spots on the cornea. Pagenstecher and Saemisch have recently called attention to the frequent occurrence of corneal spots in strabismus. * * * At least I find specks on the cornea much more common in hypermetropia with strabismus than in hypermetropia without strabismus. It is true, there may be still another connection between specks on the cornea and strabismus, to which Ruete has already directed attention: an inflammation, namely, which produces these specks on the cornea, may extend beneath the conjunctiva to some of the muscles or their envelopes, and produce, first, a spasmodic, and afterwards, a nutritive, contraction. Such cases I have already mentioned above. They are, however, comparatively rare; but they may in part explain the preponderance of specks on the cornea in hypermetropia with strabismus.

"3. *Peculiar structure or innervation of the muscles; easy mobility of the eyeballs inward.* Not infrequently a congenital insufficiency of the musculi recti interni occurs. It may readily be assumed that the opposite also may be the case; and, in fact, some eyes converge without any particular tension up to 3″, nay, even up to 2″, and less from the eye. We may assume that form and position of the eyeball exercise as much influence in this respect as the structure of innervation of the muscles. * * *

"If in a family one or two labor under strabismus convergens, we may be nearly sure that in some other members hypermetropia will occur; but that in the same family most of the hypermetropics should be afflicted with strabismus has very rarely occurred to me.

"In the highest degree of hypermetropia, strabismus is rarely observed. This need not surprise us. In such cases the power of accommodation is, even under abnormally increased convergence, not sufficient to produce accurate images, and such hypermetropics are thus led rather to the practice of forming correct ideas from the imperfect retinal images as much as possible. We have already seen that strabismus is met with chiefly in a mean degree of hypermetropia. These belong to facultative and relative hypermetropia: the eye can adapt itself for parallel and even for diverging rays, and can, moreover, maintain this accommodation for some time, yet often only with convergence of the visual lines to a point, situated closer to the eye, than the point whence the rays proceed. The minimum of hypermetropia at which strabismus occurs, depends undoubtedly on the angle λ, and on the range of accommodation; the less the latter, and the greater A is, the less degree of hypermetropia will be sufficient. But diminished energy or paresis of accommodation by itself is as little liable to produce strabismus, as is the diminution of the range of accommodation connected with the increase of years.

"Both the internal muscles of eye are therefore to be considered as shortened. The shortening, at first dynamical, has in the constant strabismus become organic; it is the result of excessive action, with relaxation of the antagonistic muscles; morbid structural change does

not exist. That both interni muscles are shortened, depends upon the habit of keeping the fixed object to the side of the deviated eye so that even in the nondeviated eye the musculus rectus internus is brought into relatively strong contraction. In this position the hypermetropia of the nondeviated eye is best overcome. Also when the strabismus has become constant, a comparatively stronger tension of the internal recti muscles is connected with the fixing of an object, whereby the angle of squinting is increased—of course in a less degree where a great angle of deviation already exists, because increased tension is then attended with less motion; after tenotomy the increase of the convergence often again appears very well marked in these cases on fixing an object. This increase of convergence on fixing, when a correct position has been obtained by tenotomy, is important in a double point of view."

We shall now review the opinions of a few writers on the accommodation theory.

Parinaud[1] says:

"At the moment when the influence of de Graefe imposes the muscular theory, the works of Donders make a happy diversion. Donders establishes the connection between squint and the errors of refraction; in the second place he shows that hypermetropia develops convergence. Unhappily he had not considered squint as a whole; he sees only one side. For the other part, he introduces into the question an element of confusion by wishing to establish between apparent squint and real squint a bond which does not exist.

"Donders has the great merit of showing again the rôle of convergence in the pathology of squint, but he studies it too much as a physician, and not enough as a physiologist. He does not conceive the alternative of convergence as a trouble of innervation to oppose the muscular trouble. He defends himself, on the contrary, against Giraud-Teulon, from having wished to lessen the rôle of the muscles. He fails to specify, to distinguish the two kinds of associated movements, those of direction, and those of convergence; he fails to show the independence of innervation to these two kinds of movements. That is the knot of the question."

Zentmayer[12] says:

"It appears to me that when one approaches the subject with an unbiased mind and considers the facts as they are, the accommodative theory accords best with these facts, and when it fails in individual cases these can well be explained by other known causes of strabismus, such as congenital palsies, structural defects or anomalies, favoring effects of abnormal orbital axes, divergence weakness, and congenital amblyopia."

Dr. Cassius D. Westcott, in discussing Dr. Zentmayer's paper, says:

"My experience is that convergent excess occasioned by excessive accommodation due to hyperopia is sufficient cause of convergent squint in a large number of cases."

Howard F. Hansell[13] says:

"Since the functional internal squint of children is indirectly dependent on hyperopia, its cure can be obtained only by relieving the strain on the accommodation induced by the effort to overcome the hyperopia.

"The convergence-center in the hypermetrope is overexercised, and its growth (together with that of the muscle) is unduly prompted, therefore hypermetropia is preeminently the favoring circumstance in the production of squint, and determines the direction of the movement of the visual axis."

Stevens[7] says:

"Turning to the anatomical side of the question, we find nothing to sustain the propositions of Donders. The more recent investigations of the anatomy of the nerve centers controlling these functions show not only that the nucleus controlling the function of accommodation is distinctly separated from that governing the convergence, but that the nerve fibers from each root pass separately out from the brain, and that they are only united within a common sheath after they have traversed a considerable space as separated fasciculae.

"Such considerations suggest that these two functions, which usually act in close agreement, so act as a result of habitual, not of organic, association.

"Training, or a necessity which interposes important obstacles to the habitual association of action, quickly enables the individual to disassociate the two functions absolutely."

Savage[4] says:

"If hyperopia were even the chief cause of the internal squint, the cases demanding operative interference would be few and far between. Hyperopia must occupy a secondary place as a causative factor of esotropia; nevertheless, it is an important place, for a removal of this cause, by the wearing of convex lenses, early in childhood, often will make the other cause or causes inoperative."

The Fusion Theory

Before studying this theory, it may be well to make a few remarks with reference to the binocular act of vision. *The essentials for binocular vision are two healthy maculae in two eyes whose motor functions are in perfect order, presided over by a healthy cerebrum.* The first essential to binocular vision is *predominating maculae;* i.e., the macula of each eye must be so developed as to demand that the image shall fall on the macula. In other words, the acuteness of vision at the macula should be several times greater than at any other part of the retina. Where this is true the retinal reflex from the macula will send in a sufficiently strong stimulus to the external muscles of the eye to force that eye into such a position that the image of the object of attention will fall on the macula, in order to secure the most perfect picture possible. The second essential to binocular vision is a normal-

functioning set of ocular muscles. This implies that they should be of proper comparative strength and under proper healthy nervous control. The movements of the eye are the most complex in the human anatomy, and its nervous mechanism is of necessity complex. The third and last essential is a properly functioning nerve center, whose duty it is to receive these retinal images, and convert them into a single mental picture, an exceedingly complex and little understood function, but none the less interesting on account of its intricacy.

In the human being there are two retinae so placed as to have corresponding parts, one with the other. The right or nasal half of the right retina corresponds with the right or temple half of the left retina, and vice versa. The macula of one eye corresponds with the macula of the other. *Fusion* demands such adjustment of the macula that the image of an object seen will fall on the macula of each eye. These images, in turn, are sent to the cortical center of vision in the brain and stimulate the same set of cells and the two images on the two retinae make but one cortical stimulus, and one impression, and only one object is seen. This uniting of the two retinal images into one mental image is called *fusion*. Any hindrance or want of proper action in this mental act is called interference with *fusion*, and the power or faculty or converting these two retinal images into one mental picture is called the *fusion faculty*. This faculty, if there be such, is innate in man. The so-called *fusion theory* of squint ascribes squint to a want of proper development of *this faculty*.

Claude Worth's theory is as follows:[14]

"In a case of convergent squint there is, in addition to the most obvious symptom, the deformity, always a *defect of the fusion faculty*, and there is nearly always a suppression of the vision of the deviating eye. In the human infant the motor coordinations of the eyes are already partially developed at birth. During the first few months of life these serve (in the absence of any disturbing influence) to maintain *approximately* the normal relative directions of the eyes. Soon the fusion faculty begins to develop. I have found distinct evidence of binocular vision in the sixth month. Normally the development of the fusion faculty is well advanced by the twelfth month, and complete before the end of the sixth year. When the fusion faculty has begun to develop, the instinctive tendency to blend the images formed in the two eyes—the 'desire for binocular vision,' as it is called—will keep the two eyes 'straight.' When the fusion faculty is fairly well developed neither hypermetropia, nor anisometropia, nor heterophoria can cause squint. In fact, then nothing but an actual muscular paralysis can cause an eye to deviate, in which case the resulting diplopia is intolerable. Sometimes, however, owing to a congenital defect, the fusion faculty develops later than it should, or it develops very imperfectly, or it may never develop at all. Then in this case, there is nothing but the motor coordination to preserve the normal relative directions of the eyes, and anything which disturbs the balance of these

coordinations will cause a permanent squint. *Thus the essential cause of squint is a defect of the fusion faculty.* In the presence of this fundamental cause the eyes are in a state of unstable equilibrium, ready to squint either inward or outward on slight provocation."

The commonest provocations to the overthrow of binocular vision are, according to Worth: (1) Hyperopia; (2) Anisometropia—marked difference in the refraction of the two eyes; (3) Hyperphoria—vertical imbalance of the muscles; (4) Esophoria and exophoria—lateral imbalance of the muscles; (5) Specific fevers. Such cases usually have considerable hyperopia, enfeebled accommodation, coupled with poorly developed fusion sense. These upset the unstable equilibrium and squint becomes apparent. (6) Violent mental disorders and convulsions, severe fright in absence of fusion faculty, upset equilibrium of convergence center. Injury during birth and occlusion of eyes for too long a period. Heredity is an influential factor. In a fair number there is a distinct history of strabismus in one or more members of the immediate family. Worth found 51.78 per cent where information was reliable.

Browne and Stevenson[15] say:

"Beginning with an undeveloped and imperfect instinct for clear definition and binocular fixation, the eye, after it has been tilted beyond the required point, is still further deprived of its education. From having an occasional, albeit perhaps a blurred view of its object, it becomes indifferently directed on space, and is, as far as its macula is concerned, unemployed.

"The outcome of the foregoing facts is to show that the *failure to accomplish binocular vision*, in accordance with our ancestral inheritance and our normal requirements, is at the bottom of squint; that failure may be due to pathological cause, as in the case of nebulae, or it may be a mere matter of faulty growth, as in hypermetropic astigmatism. Beyond this all is normal. The muscles are not at fault—*convergence is the function of the internal recti*, and that function is effected by the motor centers."

De la Hire (Javal) thought the eye deviated because the most sensitive part of the retina did not come within the prolongation of the optical axis. He says:

"It is to cause objects to impress themselves on that part of the retina that the eye takes a vicious position."

We will see that some of our American confreres are warm advocates of this theory. Linn Emerson[16] says:

"As we have a speech and an auditory center, there is no question in my mind that we have a fusion center. The presence of this center is obviously necessary for development or manifestation of a fusion faculty. But its loss or absence is not the only cause which will produce deterioration or loss of the fusion faculty.

"Worth arbitrarily divides the fusion faculty into three grades, but in reality, there are 'fifty-seven varieties' or grades, and I emphatically affirm that in all cases of concomitant squint this faculty is in some degree deficient; the structural defect, or the refractive error may be the cause of the lack of development of the fusion sense, but the deficiency of the fusion sense is the cause of squint.

"The prime cause of this variety of squint is deficiency or absence of the fusion faculty. This has been disputed by some observers who have failed to grasp the three grades of fusion described by Worth, but I can honestly say in the observation of several thousand cases covering a period of more than fifteen years that I have yet to see a case in which the fusion faculty was not absent or deficient.

"In my opinion, for lack of a better explanation, we must give the inherent desire for single binocular vision in the human being as the stimulating force over the muscle coordination powers, as a strong contributing factor in keeping the eyes parallel, certainly until the child is six months to one year of age, when there is marked association between accommodation and convergence or any fusion center developed."

Valk expresses himself thus:[3]

"My conception of this physiological act is that the movement of the eyes from one point of regard to another is a purely voluntary one, controlled by the higher brain centers, while their action in the fusion of the images according to physiological law is controlled by the lower or automatic brain centers."

He further says:

"But the strongest and most important function of the eye and the keynote of binocular vision is that of the fusion power or guiding sensation."

W. B. Lancaster[17] says:

"It is natural for the eyes to deviate. It is only when something holds them in alignment that they do *not* deviate, therefore in the *absence of this something* we have the cause of deviation. This 'something' is the desire for fusion, coupled with the ability to maintain it; therefore the absence of either the desire for fusion, or the ability to maintain fusion, or both, will cause deviation."

Duane[5] in a footnote says:

"It has seemed to the translator that the lack of ability to perform fusion in these cases is not so much the cause of squint as the result of it."

In reply to a special request with reference to the existence of a fusion faculty, Dr D. D. V. Stuart, Jr.,[18] associate professor of clinical neurology of the Georgetown University, has the following to say:

"My objection to the 'Fusion Theory' of Worth is simply this: The theory assumes the existence of a special 'fusion faculty' and a 'fusion center.' Both these assumptions seem to me unnecessary for the understanding of binocular vision.

"The points I wish to make are two: First, true fusion of the optical images of objects seen with both eyes does not take place; otherwise there would be no such thing as stereoscopic vision. We may for convenience speak of two superimposed images, seen from slightly different angles, as fused; but, of course, they are not. Second, it is my belief that we have a constant diplopia for objects within the field of vision but not directly focused on; and that we habitually disregard this, just as a squinter learns to ignore the image seen with the deviated eye.

"Because of these two points, I do not see why we need assume any special faculty or center for 'fusion.' It seems to me adequate to think only of a center for convergence, allowing the two eyes to bring a single object into range with the point of greatest visual acuity of each retina. Irritative lesions of the convergence center would account satisfactorily for cases of nonparalytic squint, and destructive lesions for divergent cases."

The weakest point in the *fusion theory* of Worth is that most cases of concomitant squint develop in children after they are three years old and have enjoyed binocular vision for two or three years. If it were a defect or fault of fusion entirely, it seems that we should find our squinters during the first years of life instead of waiting three years for it to *develop.* It does not seem logical that a congenitally defective fusion faculty would work for two or three years and then *break down.* It would be more reasonable to suppose that with each day of binocular vision that faculty would certainly increase and grow stronger and by the third or fourth year be strong enough to overcome all obstacles except, as Worth has said, "paralysis of an ocular muscle."

The very fact that 25 to 40 per cent of these cases can be cured by use of glasses, treatment, and exercise, and perfectly good binocular vision obtained would seem to me to disprove a specially defective *fusion faculty.*

It is also difficult to believe that the exercise of the fusion faculty for ten to twenty minutes at a time daily for a few weeks could so improve a defective fusion faculty as to make it function properly ever after. We do not accomplish any other mental feat with such ease.

It would seem that it is *not so much a defect* in the fusion faculty as it is a *disturbance or want of harmony* in the proper uniting or coordinating of the several stimuli, or retinal reflexes, sent in to the centers for convergence. A given object creates a retinal image on each retina. The dominating maculae of good eyes send a message to the convergence center to set each eye in such a position that the image shall fall on the retina of each eye in order that it shall be properly *fused.* At the same time the accommodation center, which is allied but not identical with the convergence center, records a stimulus to focus that object and obtain the most perfect image possible.

Now, squint is due to a disturbance of this complex act. It may be a fault in receiving the image, that is, in the retina; it may be a fault in transmitting the stimulus to the cerebral centers, or it may be a fault in receiving the stimuli and in turn transmitting it into the proper motor stimuli. We are practically all agreed that we have an excess of convergence, but we are not agreed as to what causes that excess. The muscle men say it is too strong a muscle, the fusion men say that it is a defect in the fusion faculty, and the advocates of Donders' theory say it is due to hypermetropia. In answer to the muscle men we should say: "The muscle is not at fault, because there is no limitation in movement in recent cases, the eye straightening in sleep and under anesthesia, and many cases are cured with glasses which have no effect on the muscles." To the fusion men we should say: "The eyes are straight for three or four years and then cross. Why does fusion break down after three or four years' use?" Of the Donderites we should ask: "Why do not all hyperopes squint? The majority do not and often the worst do not; besides, many cases of squint have no hyperopia or very little, and even some cases of myopia are internal squinters."

The Nervous Theory

With the three theories already advanced, it might be said to be superfluous to introduce a fourth, but the study of the literature on squint is so replete with writings on the influence of the nervous control, or want of control, in squint, that I feel justified in introducing this theory, disclaiming at the same time any originality for it.

This theory should be divided into two general heads: *Pathologic nervous lesions. Functional nervous manifestations.*

Pathologic Nervous Lesions.—It has been known from the very earliest times that paralysis caused squint. In fact, that was the dominant idea of squint etiology for centuries. Frank paralytic squint will be discussed under a separate chapter, and the remarks following will not deal with paralysis, but with results of paralysis, or disease.

R. I. Lloyd[19] is very much impressed with the number of permanent squint cases he has found with pathologic lesions. He says:

"It is our belief that there is a pathologic cause for strabismus.

"Group 1 should include all cases due to loss of Foveal Guide.

"Group 2 would include all cases due to Muscle Paralysis.

"Group 3 would include all cases due to Positive Scotoma (blurred image).

"Group 3 cases can pass on to blindness and to Group 1; and a Group 1 case might, now and then, emerge from that unfavorable class and end in Group 3, or perhaps recover.

"Loss of the Foveal Guide need not be more than an absolute defect of the central one degree to allow the eye to deviate. Total

blindness is not necessary to bring about this state. It should be noted that every absolute defect is surrounded by a zone of relative defect and that this one degree absolute defect means absolute blindness of the all important fovea with a surrounding zone which is below par.

"Deviation (page 110) in or out regularly goes with traumatic macular disease, coloboma of the macular area, chorioretinitis involving the central zone, etc. All of the foregoing remarks apply only to other than alternating deviations. None of the suggestions which apply to convergent or divergent squint of the unilateral variety is of use in considering *alternating* squint. This variety has usually good vision for each eye and it is reasonable to look upon this very peculiar and stubborn deviation as an exclusively cortical or nuclear abnormality, probably due to malformation or eccentric nerve habit.

"It is our opinion, then, that strabismus is associated with pathology, and is not a nonuse complex. For this reason, cursory examinations of squint cases are out of place. More accurate measurement of deviations are in order and can be done without consuming the time usually thought necessary to accomplish this. More fields of these cases need studying and neurological assistance is frequently of great value to clear up the case. A technique which will more certainly point out the old paretic muscle cases is needed, as otherwise operative results will disappoint."

It has been Lloyd's observation that a larger percentage of constant *squinters* have defective visual fields, both in the good eye as well as in the deviating one, with diminished visual acuity in each. He presents this as evidence that there has been previous etiologic nervous pathology. His findings are of great importance and should lead all ophthalmologists to more carefully study his squint cases.

It has been frequently observed that a certain number of squint cases follow paresis or paralytic lesions which occurred early in life and had left no trace. Of these cases, Javal, Duane, Maddox, Harman, and many other writers have written extensively.

These cases usually occur early in life and are associated with marked amblyopia, even where no pathology can be found. Duane has classified a certain number of these cases under *congenital squint.*

Functional Nervous Manifestations.—Functional nervous manifestations in etiologic relation to squint have been recognized for more than a century. Mackenzie[20] in 1855 gave a fairly definite statement of the functional nervous theory. He said:

"The cause of squint should be sought elsewhere than in the muscles of the eye, elsewhere than in the retina; that is to say, in the brain and the nerves, organs which preside over the association of the acts of the muscles of the eyes."

Then, alluding to the first attempt of Dieffenbach and others to cure squint by myotomy, he adds these significant words:

"It ought not to be astonishing, then, when one comes for the first

time to the idea of several doctors, that division of the adductor muscle can prove useful in convergent squint, this idea of correcting the disturbed exercise of a nervous function by section of one of the muscles the acts of which are disturbed because of this nervous disorder, has been received with suspicion, then abandoned."

After Mackenzie, Javal for a third of a century devoted much effort in elucidating the relation that existed between comitant squint and the nervous system.

The first use of the excessive "innervation of convergence" as a neurocausal factor in the production of squint was made by Krenchel in 1873. About the same time Hansen Grut read a paper in New York in which he advocated the functional nervous disturbance of convergence as a cause of squint. After him we find Mules and Berry supporting the same idea.

Possibly no one has given more study to squint than Parinaud,[1] and certainly but few have better stated its etiology. His ideas comprise all of the good in the so-called Worth's Fusion Theory, as well as much in the accommodation theory of Donders, and he is at the same time the chief advocate of the nervous theory. I shall now quote him at length:

"It would be a new error to see in squint only innervation of convergence. One can understand its pathology only with a larger conception which permits synthetizing the diverse influences which enter in. I have defined squint: *a fault of the development of the apparatus of binocular vision, bearing at the same time on the motor part and the sensory part of that apparatus.*

"The motor part is represented in convergence. The immediate *cause of the squinting deviation is always a trouble of innervation of convergence.* The two principal factors of convergence are the *retinal reflex of convergence and accommodation.* All the causes act on the convergence through these two intermediaries. These causes have the common characteristic of constituting an obstacle to binocular vision; they act more efficaciously when the subject is young; that is to say, when the apparatus of binocular vision is less developed.

"In order to understand squint, this conception is still not enough; it does not suffice to consider the causes which determine the squint, and the mechanism by which they act on convergence. It is necessary to consider the secondary modifications which are produced and which, insensibly, change the character of the squint. Excess of innervation of convergence, which at first characterizes convergent squint, tends to disappear in the measure that the apparatus of binocular vision changes; that is the cause of spontaneous correction of the eyes in certain cases. More often the deviation persists in spite of the disappearance of excess of innervation which caused it. It is then maintained by the retraction of the periocular tissues, of the capsule of Tenon in particular. This retraction caused by the deviation becomes in its turn the cause and radically transforms the character of the squint by a process which is found in other deviations of nervous origins.

"The sensorial part changes at the same time. The modifications represent the alteration of the three fundamental properties of the apparatus of binocular vision, namely, the retinal reflex of convergence, diplopia, the manner of projecting the retinal images.

"This conception of squint permits one to establish unity in the midst of diverse influence which produces it and of the numerous clinical forms which it takes.

"I have noted the fact that convergent squint is at first characterized by an excess of innervation; the cause of the squint is not in the surroundings of the eye, but in the brain. If one observes this squint at the age of twenty, it is entirely changed. The excess of innervation of convergence has disappeared; it is even replaced, usually, by the complete abolition of this same innervation. The deviation is maintained solely by the retraction of the periocular tissues. In spite of the similarity of symptoms, it acts, from a surgical point of view, from two different affections. How can one expect the same operation to produce the same effect in these two extreme cases, or in the intermediary forms? There is still evolution of squint, which modifies the immediate effects of an operation, to consider, evolution which will be different according to whether retraction of the periocular tissues exists or does not exist, or according to whether one operates on convergent or divergent squint."

Sauvineau[21] has the following to say with reference to Parinaud and his theories:

"But the theory of innervation of convergence remained an indefinite idea until Parinaud established clearly the existence of a special innervation, foreseen by the previously named authors

"In a work on associated paralyses and in several later publications, he opposed to paralysis of the ocular muscles, *paralysis of function,* aiding the muscles in certain actions and inhibiting them for others. By the facts of paralysis and of contracture of convergence, he showed especially that innervation of this function is susceptible of being altered independently of that of the associated movements of direction. (p. 142.) This point established, the reasoning by which de Graefe and Giraud-Teulon have tried to exclude the influence of innervation in the pathology of squint, fail of themselves. Innervation of the associated movements of direction is, indeed, intact; it is that of the movements of convergence which is altered.

"In squint, in fact, it is only innervation of convergence which is to be considered. In spite of the divergence of the optical axes, the associated movements are conserved in the lateral direction of regard, for the innervation of the muscles is intact for the movements.

"Convergent squint is determined by an excess of innervation of convergence, a kind of contracture. Divergent squint is, on the contrary, characterized by a progressive relaxation of that same innervation, similar to paralysis, with the difference that it acts, not by accidental lesion of a nervous center, but by an error of development of apparatus."

Delogé[22] says:

"Thus strabismus, which was, according to savants, a local affection of visual or muscular origin, became, according to Parinaud, an

affection of the central control. It appears to be not only the most natural, but the only probable, explanation of certain forms of strabismus, more particularly those which supervene when optical causes are at fault * * *."

Simpson[23] says:

"In any case, his (Parinaud's) definition is extremely explicit. It is a question of a 'flaw in the development of the apparatus of binocular vision, preventing the convergence of the eyes upon a fixed object.' It is a disorder of central control on the reflex of the convergence. If this last be exaggerated, there is convergent strabismus; if it be lessened, divergent strabismus supervenes. The cerebral trouble itself may be primitive, and may by that fact prevent binocular sight, or it may supervene early and impede normal development."

C. Sauvineau[24] introduced a unique theory of the cause and treatment of squint, which supports the functional nervous theory as follows:

"The essential disturbance with strabismus is inability to focus the lines of vision on the object, and this, he insists, is the result of an anomaly in the perceiving centers in the brain. Only the visual image from one eye alone is perceived. The cross-eyed are cerebrally blind of one eye. Both eyes may be visually perfect, but the brain perception is monolateral; the brain neutralizes or drops out the image furnished by the deviated eye."

The following is from Browne and Stevenson:[15]

"The essence of squint, then, is a failure on the part of the peripheral sense-organ to inform its cerebral centers of its requirements. The muscles are not at fault, nor their motor centers; indeed, the maintenance of convergence (and accommodation) is evidence of their integrity."

Bishop Harman[25] says:

"That a defect of cerebration, involving a weakness or possible absence of that most human desire for binocular vision, is at the basis of manifest squint is, I take it, a matter of general agreement."

E. Maddox[26] has the following to say in regard to the etiology of squint:

"The great majority of convergent squints are nonparalytic, and therefore, comitant, being purely due to excessive activity of the converging innervation.

"Many cases of concomitant convergent squint disappear under chloroform, showing that the internal recti are not contracted or structurally altered, but only unduly innervated. In most cases this activity was at first occasioned simply by association with excessive accommodative effort called forth either by hypermetropia or possibly in a few cases by paresis of the ciliary muscle, as suggested by Javal. Convergent squint of the kind under consideration nearly always

commences about the age of three years, when children first begin to regard small objects attentively. Possibly at this age accommodation begins to require a greater effort than before, from changes in the consistency of the lenses. Or it may be that at the age when a squint begins the insulation between the two paths of nervous impulse to accommodation and convergence is still imperfect so that strong accommodation is impossible without equally strong convergence. When once formed, a squint persists from innervational habit."

Le Fever[27] says:

"Since clear vision is the chief function of the eye, the accommodation element of the stimulus predominates and too much stimulus is given to the convergence. This would cause the optic axes of the two eyes to cross and a squint to occur in all hyperopes were there not some means by which the patient can compensate for the excess convergence. *This he does by sending as much excess stimulus over the sixth nerve to the external rectus muscle as there is excess convergence, and this eye is drawn tautly back into its suspensory ligament, giving the deep-set eye characteristic of hyperopia. Or the patient may be able to separate the two stimuli and converge less than he accommodates.*"

Savage[4] also supports the nervous theory of squint in the following sentence (l.c., p. 8095) in which he says:

"The conditions causing the error exist from birth, and only one of the several natural causes undergoes any change at any time after birth. In the greater number of cases it is the character of the nerve impulse to the ciliary muscles that causes overstimulation of the third conjugate innervation center, which overstimulation causes an actual deviation, when previously there was only a tendency toward deviation—a conversion of an esophoria into an esotropia."

Dunnington[28] says:

"A lateral strabismus may result from:

"1. A paresis or spasm of one of the extraocular muscles.
"2. An anomaly of divergence.
"3. An anomaly of convergence.
"4. A combination of any two or all three of these conditions."

Duane[5] has taught us that most lateral deviations begin as an anomaly of convergence or of divergence, and that having started in this way, they gradually become constant through a secondary involvement of the originally unaffected power Worth,[14] on the other hand, contends that the deviation is the direct result of a defective power of fusion. It is possible that both factors enter into the production of a squint, but I believe the predominant cause to be a dysfunction of the converging or diverging power.

It has been evident to the author for a number of years that strabismus in the very young is due in a large percentage of cases to a general nervous instability.

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The fusion theory of Worth is but a minor part of the nervous theory. In fact, the accommodation theory of Donders is comprised in this theory when it is viewed as an *excess of innervation of convergence.*

Muscular anomalies, hypermetropia, the heterophorias, or even a moderate amount of amblyopia, would not cause squint in the presence of a well-balanced nervous system.

I have yet to see a child with functional strabismus who did not show some general nervous instability in addition to the crossed eye. If one examines into the family history of squint cases, one or both parents are neurologically deficient in some respects. Here we see our heredity playing its part on the constitution, if it is not expressed in a crossed eye.

Probably no one has written so complete and masterful a description of the disturbances of the motility of the eyes as is found in Duane's translation of Fuch's sixth edition. Certainly no one in America has done more original work on this subject, nor has any one presented the facts more precisely and concisely than the lamented A. Duane. The etiology of squint is so well given that I take the liberty of quoting it at length (p. 781):

"An esophoria may disappear if the conditions causing it are abolished or offset. Thus an accommodative convergence-excess usually disappears if the causal hyperopia and astigmatism are corrected early enough and it disappears in presbyopes who no longer accommodate excessively because they cannot. Again, in some cases a convergent squint disappears in youth, because in the growth of the face the orbits get more divergent so that the interni work with more difficulty than before (Weiss).

"In other cases an esophoria increases. This is seen in the *development of a convergent squint,* which usually begins as an accommodative convergence-excess. The steps in this development are as follows:

"(1) A child with a good deal of hyperopia or astigmatism, as soon as he begins to use the eyes much for near work, develops a varying, evidently more or less spasmodic esophoria (convergence-spasm, see supra) which is marked for near when his convergence is called into play, but which is little or not at all marked for distance.

"(2) Little by little the deviation increases until binocular fixation for near becomes impossible, and the periodic esophoria is transformed into a periodic *convergent squint.* This at first is intermittent and occasional, afterwards more and more constant. There is now more or less esophoria for distance also, although always less than for near, and there is still binocular fixation for distance.

"Careful tests at this stage will show in nearly every case that *the squint is associated with diplopia.*

"(3) After this condition has lasted for a long time, perhaps some years, the patient gives up binocular fixation for distance, so that he now has a convergent squint for both distance and near (*continuous*

squint). The deviation as measured by the screen, however, is still much more marked for near than for distance. Diplopia can still usually be elicited by the tests. The condition is still mainly one of convergence-excess.

"(4) Later still, the amount of deviation for distance begins to increase until ultimately the squint for distance equals that for near points, and both are absolutely greater than they were. This change is due to the superadding of a divergence-insufficiency to the primitive convergence-excess; the conditions, now, in fact, are those shown in the table as obtaining for a convergence-excess and divergence-insufficiency combined. The rotation of the eyes is still normal in amount, indicating that the muscles themselves are as yet unaffected.

"At this stage, diplopia, if it can be elicited at all, no longer obtrudes itself upon the patient's notice nor causes confusion. In many cases it cannot be elicited by any tests, suppression of one of the images having occurred (see p. 763).

"(5) Later, secondary muscular changes (contracture of the internus, stretching and weakening of the externus) take place in the squinting eye, producing an absolute reduction in the rotation outward and excess of rotation inward.

"In some cases it seems that a *convergent squint may develop from a divergence-insufficiency.* The steps in this case are, first, an increase of the esophoria for distance, then the superadding of a convergence-excess producing an esophoria for near. The result is, first, a periodic convergent squint (esotropia for distance, esophoria with binocular fixation for near); later a continuous squint for all ranges. Until late in their course such cases are distinguished from those starting with a primitive convergence-excess by the fact that the deviation for distance always exceeds that for near."

On page 785 he further states:

"As seen from paragraphs 684 and 685, a convergent squint usually develops out of a simple convergence-excess to which is added a divergence-insufficiency; and a divergent squint develops out of a simple convergence-insufficiency to which is added a divergence-excess or out of a divergence-excess to which is added a convergence-insufficiency."

The following excellent discourse on the etiology of squint was sent me by Professor Van der Hoeve[29] of Leyden, Holland:

"Every human being is born with a certain amount of static strabismus, because the conditions of our eyes are not symmetrical.

"If, in looking at a distance, one eye looks straight forward, it would be accidental if the other eye takes a direction of the fixation line of the first eye. To cause such a position, the conditions of both eyeballs must be absolutely symmetrical, and that is not the case. The orbits of both sides are not symmetrical, neither are the eyeballs, the muscles, the orbital fat, or ligaments. And even if by chance, the position of the eyeballs was symmetrical by fixation on distance, it would not stay so on account of movements of the eyeballs as well as side movements and convergence, because if one eye gets a certain impulse to fix on an object, it is highly improbable that this same im-

pulse would fix the other eye on the same object; then the different resistance, which the eye movements have to conquer, is in both orbits not the same, and so the same impulse will not give the same movement of both eyeballs.

"But even without voluntary movements an accidental symmetrical position of the eyeballs cannot stay, because the static conditions of the orbit change every moment, the orbital fat can change in quantity. It is decreased by illness, by hunger, by fatigue, and comes back on recovery; this does not go on symmetrically; the quantity of blood in the orbit changes with every position. Consequently there exists in every human being static squint, changing its amount and even perhaps its direction from moment to moment in consequence of the ever-changing static conditions and yet most of the human beings do not squint, therefore an apparatus is necessary which corrects this static squint and another apparatus which warns that strabismus is present.

"The normal human being does not squint because beginning squint means diplopia, and the normal human being has a great fear of diplopia; it makes one uncertain, dizzy, causes headache, etc., therefore, one will with all his forces try to get away from those double images. This horror of diplopia, which we call diplopia-phobia, is the condition which warns us that we are squinting and puts in action the correcting apparatus, the sensu motoric apparatus of our eyes. Everyone who has diplopia-phobia will try to get rid of the double images.

"We can do this in two ways, either by bringing them together or by excluding them. The first way will be followed when he has *fusion tendency* and when the squint angle is not large. The second, if there is no fusion tendency or if the angle is too large to bring the images together.

"If it is impossible to bring the images together, one of them must be excluded, and this is very difficult, especially when the images are close together and attract too much attention, hence the squinter will try to move them farther away from each other, so that he can exclude one of them. Therefore he tries to turn away one of his eyes to bring the double image in such a place that he can exclude it; we can say that his eye flees from the double image, so that he adds to the static squint which he cannot conquer, a *flightstrabismus*. So we can build up this scheme of persons, who have congenital static squint.

"In people who can bring the double images together and do this a long time, the tissues can get used to it in such a way that they get what we call *orthophoria*. Real orthophoria is impossible because the static conditions change every moment, so that also the people with so-called orthophoria have to correct this change with their sensumotoric apparatus and use the warning signal of the diplopia-phobia. Orthophoria is really heterophoria with the squint angle so small that we do not perceive it by our examination.

"If the tissues do not get well accustomed to the conditions then we get *heterophoria*.

"Donders taught us that there is a narrow connection between refraction and strabismus. A hypermetropic person has to accommodate much for infinity; a hypermetropic man with 4 D hypermetropia

has to accommodate for distance 4 D without converging; if he tries to fix on one meter he has to accommodate 5 D and to converge only one meter angle; on one-half meter 6 D and 2-meter angles, etc., always a difference of 4 units between accommodation and convergence. Now, if people always converged as much as they accommodated, every hypermetropic person would get convergent squint. This strabismus would not be concomitant, since the angle would be not the same when the patient looked at a distance or looked near.

"Happily, the connection of accommodation and convergence can be loosened and most hypermetropic people can hold their squint latent. Others who cannot do this begin to squint. If they followed convergence with their accommodation, all people with the same amount of hypermetropia would have the same squint angle. We know this is not the case. Why not? Because if anybody squints he will try to get rid of his double images by exclusion, and will bring one eye in such a position that he can exclude; he flees from the double image and adds to the refraction-squint a flightsquint. So we can explain why people with the same amount of hypermetropia have different amounts of strabismus. This explains also why correction of refraction does not always give correction of the refraction squint.

"If, for instance, a man has 15 degrees refraction squint and adds to this 15 degrees of flightstrabismus, then he squints 30 degrees; if we correct the refraction only, the refraction squint becomes corrected; if we do this, the first time after the patient squints, the flightstrabismus will vanish too; if we wait too long, the tissues have gotten accustomed to the wrong position, correction of refraction corrects only the refractive squint, not the flightsquint, and the patient will hold a certain amount of squint or try again to find a position in which it is easy for him to exclude."

NOTE: ("I use two terms which as far as I know are new in ophthalmology; these are diplopia-phobia and flightstrabismus.")

Contributing Causes of Strabismus

The following are the chief contributing causes of squint:

1. Hypermetropia.
2. Heterophoria.
3. Anisometropia.
4. Amblyopia.
5. Structure and shape of the orbit and the eyeball.
6. Declination.
7. Acute illnesses.
8. Injury at birth.
9. Heredity.
10. Miscellaneous causes.

Hypermetropia.—Since Donders called the attention of the world to the etiologic relationship of hypermetropia to squint in 1864, that relation has been practically universally recognized. He saw in it,

however, the sole cause of squint, which latter idea has not been generally accepted. Worth[14] says:

"It will thus be seen that the degree of the refractive error has very little to do with the question of whether the patient shall or shall not squint in the first instance, though, of course, when the squint is once established, the refractive error becomes a very important factor." (p. 52.)

Worth first denies the etiologic relationship of refraction to squint and then in concluding his remarks, admits it.

Browne and Stevenson[15] say:

"Therefore, hypermetropia is preeminently the favoring circumstance in the production of squint, and determines the direction of the movement of the visual axis."

The mode of action of hypermetropia in contributing to squint is as follows: By nature we have learned to use and associate accommodation and convergence, and an excess of accommodation calls for an excess of convergence. When a person with normal eyes looks at a distance, there is no need for convergence or accommodation, but when his eyes are called upon to look at an object one meter distant, both accommodation and convergence are called into action. He uses one D. of accommodation and converges one meter angle on looking at an object one meter distant, and on looking at thirteen inches (the reading distance), he accommodates three D. and converges three meter angles. Now, suppose we have a case with three D. of hypermetropia. This patient must accommodate three D. to see at a distance and at the same time, if the relations of accommodation and convergence were inflexible, the eyes must converge three meter angles. Now, for looking at thirteen inches the patient must accommodate six D., and that would call for six meter angles of convergence, which would direct the eyes to six and two-thirds inches, and this would cause the two images to fall outside the fovea and thus produce blurred images. In the interest of acute vision, one eye is directed so as to receive the image on the fovea, while the other is permitted to turn in and receive the excess of convergence. This is the theoretical explanation, but, fortunately, facts and theories do not always coincide. If they did, probably most of the hypermetropes would squint, and this is not true. The reason is that the relation between convergence and accommodation is not a cast-iron one, and accommodation and convergence are sufficiently disassociated to permit the hypermetrope to see without squinting, besides it is quite probable that an extra stimuli is sent in to the abductor muscles so as to prevent an excess of convergence. (See le Fever.)

The very fact that so many cases of squint are cured by the use of glasses is sufficient proof to show the etiologic relation between hypermetropia and squint. Claude Worth says (l.c., p. 53):

"Though hypermetropia stands in some close etiological relation to convergent squint, it is not the essential cause of the anomaly."

Heterophoria.—Since the essential for binocular vision is two well-defined retinal images formed on two healthy maculae, it can be readily seen how anything which would prevent these images from falling accurately on each macula may aid in the production of squint. In heterophoria, if one eye fixes accurately, the other eye tends to turn in, out, up, or down, depending on the amount and character of heterophoria present. Esophoria of 10 to 15 degrees no doubt aids in determining convergent squint in many cases, especially if to such a case one adds 2 or 3 D. of hypermetropia and particularly if this is combined with astigmatism. This combination of contributing causes may readily determine whether a given case shall squint or not. Hyperphoric defects are less readily overcome than the lateral defects. This can be explained from the fact that a No. 2 or 3 prism base up or down will produce diplopia, whereas, a No. 10 to 15 degree prism base out may be readily overcome, or a No. 6 to 8, base in, will usually be as quickly neutralized; therefore, hyperphoric defects cause a marked blurring of the two images and may act strongly toward producing squint. Exophoria has a tendency to the production of external squint only.

Anisometropia.—Anisometropia, or unequal refraction of the two eyes, acts by interfering with the image of the retina. Browne and Stevenson[15] say:

"So long as images of equal dimensions and equal sharpness are brought in by the refractive media, so long will the greatest degree of comfort be reached by receiving these images of the maculae."

In these cases one of the images is usually nice and clear, whereas, the other image is very much blurred and defective or entirely too small to correspond with the fellow image. Hence, the call for fusion of these dissimilar images is markedly lessened, thus permitting the eyes to wander. Buffon in 1743 was first to call our attention to the fact that unequal strength of the two eyes might cause squint.

Amblyopia.—Amblyopia, from whatever cause, would naturally interfere with the receiving of clear retinal images and in turn hinder the proper stimulus to binocular vision. Unilateral amblyopia is usually the character of amblyopia which we find in these squint cases. Amblyopia may be congenital or acquired. Congenital amblyopia,

strictly speaking, is fortunately a very rare affection. Worth[14] says (p. 62):

"In the course of examining the refraction of many thousands of patients who have never squinted, up to the end of 1902 I only met with twenty-three cases of amblyopia of 6/18 or higher, which I felt justified in regarding as congenital."

In these cases there is no fundus lesion present, and the field of vision is normal. The defects seem to be a defect in the maculae. There is a want of a dominating macular region. The vision of the maculae is about the same as that of the surrounding retina. The vision in congenital cases ranges from 6/12 to 6/60. Worth claims he never saw a case of congenital amblyopia where the vision was worse than 6/60. We might have an amblyopia from scars on the cornea, disease of the iris, congenital cataract, central retinal disease, or disease of the nerve. Anything which might produce decrease in the visual acuity in one eye might readily interfere with the innate desire for binocular vision and thus aid in the production of squint.

Structure and Shape of the Orbit and the Eyeball.—Donders[11] called our attention to the fact that the position of the eyeball in the orbit and the shape of the eye being short antero-posteriorly may make it more easy for the eye to turn in under a given stimulus. Many (Reber, Duane, et al.) think that the peculiar inclination of the orbit, together with the shape of the eyes and possibly an advantageous insertion of the internal recti, has much to do with the production of squint.

Declination.—We are indebted to Dr. George T. Stevens[7] for calling our attention to this as a factor in the production of squint. He was so imbued with the idea that declination was a determining factor in the production of squint that he devised operations for its relief and reported many favorable results. His son, Dr. Chas. Stevens, is still a warm advocate of his idea. There is no doubt but that a marked declination would seriously interfere with the proper reception of the two macular images and hence interfere with binocular vision, and thus act as a cause in the production of squint.

Acute Illnesses.—It has been recognized that squint follows many of the acute illnesses, such as whooping cough, measles, scarlet fever, and acute condition associated with convulsions. A majority of the parents on bringing the child for consultation will state that he was all right until after some specific acute illness. The mode of action in these illnesses is probably as follows: The child's general condition is below par, and the nervous system is suffering from both the fever and the toxemia from specific infection present. He is given something to play with or something to read, and in making an effort to see

distinctly with an already weakened accommodation, the extra effort probably upsets the delicate nervous relation between accommodation and convergence. There are usually present in these cases other contributing causes, such as hypermetropia or astigmatism. The combination of contributing causes upsets the relation between accommodation and convergence, and the child squints. In some of the cases in which there were convulsions there is probably a paresis of one of the rectus muscles, and by the time this is relieved the habit of crossing the eyes is established.

Injury at Birth.—These usually act by producing a paralysis or a paresis of the external rectus muscle and probably prevent the normal motor coordination (Worth) or the guiding sensation (Savage) from keeping the eyes straight until fusion or the natural desire for binocular vision has developed. By the time the paralysis has been relieved, the desire for binocular vision has been lessened and squint now prevails.

Heredity.—There is no doubt but there is quite a marked hereditary tendency to squint in many cases. I have often seen it run through several generations. However, Donders[11] says:

"Moreover, I have not been able to satisfy myself that a special tendency to strabismus may be hereditary. Let me be understood. In a very high degree hypermetropia is hereditary. It is a rare thing, with hypermetropia structures of the eyes in one of the parents, not to find hypermetropia in some of the children, but whether this hypermetropia in the parent was combined with strabismus or not, has, if any, certainly only the slightest influence on the hypermetropic children born of them."

Notwithstanding these remarks, the statistics of Claude Worth are so convincing that it leaves little doubt. Worth[14] says (p. 59, l.c.):

"Hereditary influence is a marked feature in any series of cases of convergent squint. In 1,373 cases of squint in which I was able to get pretty reliable information, there was a history of squint in parent, grandparent, brother, or sister of the patient in no less than 711, or 51.78 per cent."

It has been my observation that heredity has its bearing on practically every case. This is not always expressed by a crossed eye in one of the parents, but it may be expressed in a general unstable nervous system.

Miscellaneous Causes.—Under this heading we might mention fright, irritating effect of intestinal parasites, malnutrition, general nervousness associated with one or more of the already mentioned causes of squint. Such attributed causes as looking at a curl, a spot on the nose, or the imitation of some one else are not of sufficient importance to deserve more than mention.

The following interesting remarks on the etiology of strabismus were received from E. Jackson just as this volume was going to press:

"The causes of strabismus include:

"Imperfect development of the coordinating mechanism of binocular vision.

"Interference with coordination by abnormal accommodation, excessive or deficient (hyperopia or myopia).

"Reversion to the monocular vision type with diverging visual axes.

"Paralysis and paresis of the extraocular muscles, or mechanical interference with them.

"Worth's fusion theory is useful, but few cases are relieved by the use of fusion training without other measures."—(Personal communication from E. Jackson.)

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CHAPTER III

ANATOMY OF THE ORBIT AND OCULAR MUSCLES

It is essential for the student of squint to have at least a good working knowledge of the anatomy of the ocular muscles and the orbit. In this chapter we shall present only an elementary anatomical outline. For more complete information on this subject he is referred to the various texts discussing it. The following description is taken from the American Encyclopedia of Ophthalmology, the Encyclopedie d'Ophthalmologie Francais, and the writings of Stevens,[1] Fuchs,[2] Motais,[3] Dwight,[4] and Duane:[5]

The Orbit

The orbit is the bony cavity in which the eye is located. The bony rim about the orbit is very close, firm bone. This protects more securely the delicate organ which it contains. The orbital cavity is more or less of a cone with its apex behind and its base or overture forward toward the general plane of the face.

The orbital cavities are more or less quadrilateral in shape. This is particularly true in their anterior aspect, and more marked in some skulls than in others. These two cone-shaped cavities approach one another at their apices, and if prolonged, would intersect about two centimeters behind the chiasm. They each form an angle with the sagittal axis of the face of about 25 to 27 degrees. Dwight[6] gives the angle between the two orbital planes as 42 to 45 degrees. The orbital cavities are inclined downward and outward, forming an angle of about 15 degrees from the horizon.

According to Stevens,[7] the various depths of the average adult orbit, measuring from the optic foramen are as follows:

To the internal angle at its base, 45 mm.
To the external angle at its base, 50 mm.
To the middle of the superior arch, 52 mm.
To the middle of the inferior arch, 52 mm.

The orbital cavities are much deeper on their medial (nasal) sides than on their lateral (temporal) sides, as shown by Figs. 5 and 6 taken from Fuchs (p. 713):

The several sides of the orbit are spoken of as the roof, the medial side, the lateral side, and the floor.

A number of the bones of the face enter into the construction of the orbit. Stevens[7] describes the orbit as follows:

"Thus the roof, the largest of the four walls (paries superior), is formed anteriorly by the orbital plate of the frontal bone, and more

posteriorly by the lesser wing of the sphenoid. Below, the orbital process of the superior maxilla, the orbital process of the malar bone, and the orbital process of the palate bone constitute in their order from before backward the floor of the orbit (paries inferior). The wall of the nasal side (paries medialis), the least in extent of the four, is formed mainly by the os planum of the ethmoid and the orbital surface of the lachrymal. From before backward the four bones entering into the composition of this wall are: the nasal process of the superior maxillary, the os unguis, the os planum of the ethmoid, and a small part of the body of the sphenoid. The nasal process of the superior maxilla constitutes a portion of the inner anterior wall

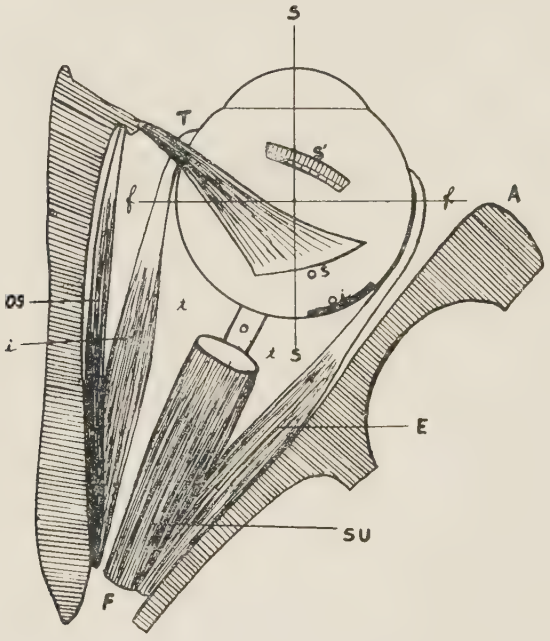

Fig. 5.—Horizontal section through the orbit (schematic). The apex of the orbit is formed by the foramen opticum *F*. The external wall of the orbit does not reach as far forward as the internal, so that its anterior margin, *A*, lies in the same plane as the equator of the eyeball. At the anterior extremity of the internal wall of the orbit is shown the trochlea, *T*, which in reality lies in the upper and inner angle of the orbit, and hence would not be met with in the cross section of the orbit that is here represented. The external rectus, *e*, and the internal rectus, *i*, bound the muscular funnel, *tt*. A portion of the superior rectus (*su*) is cut away to show the optic nerve, *o*. *S'* is the insertion of the superior rectus upon the eyeball lying oblique to the margin of the cornea. *os'* is the fan-shaped insertion of the tendon of the superior-oblique, *os*; *oi* the line of insertion of the inferior oblique; *ff* is the frontal, *ss* the sagittal, axis of the eyeball. (From Fuch's *Textbook of Ophthalmology*.)

as well as a portion of the anterior floor. The lesser wing of the sphenoid with its optic foramen forms the posterior portion, not only of this, but of the other walls.

"The greater wing of the sphenoid and the malar bone, with the temporal extension of the orbital portion of the frontal bone, constitute the elements of the exterior wall (paries lateralis)."

At the base of the orbit are a number of foramina through which very important vessels and nerves pass. Among these is the optic

foramen, which transmits the optic nerve and the ophthalmic artery. The next most important opening is the superior orbital fissure which lies vertically, anteriorly, and externally to the optic foramen. This fissure transmits the third, fourth, ophthalmic division of the fifth, and the sixth nerves, and the ophthalmic vein. In the edge of the superior orbital fissure is to be observed a tubercle to which is attached the common tendon of several of the ocular muscles.

The inferior orbital fissure, located in the lower external orbital wall, transmits the maxillary nerve with its zygomatic branch, the infraorbital vessels, together with one or two small veins. There is a

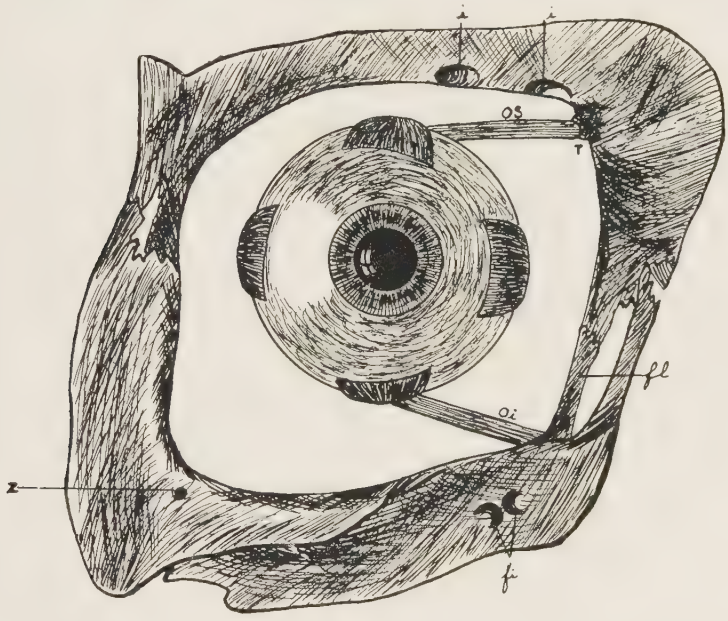

Fig. 6.—Anterior orifice of the orbit with the eyeball. The tendons of the four recti muscles are cut off near their insertion upon the eyeball, but the inferior oblique, *oi*, and the tendon, *os*, of the superior oblique are left entire. The latter comes out from the loop of the trochlea, *T*. To the temporal side of the trochlea lies the supra-orbital notch, *i*, and somewhat to the outside of this there is a foramen, *i'*, which is not regularly present, for a branch of the supraorbital nerve. In this case the infraorbital foramen, *fi*, is also abnormally divided into two distinct foramina. *Z* is the orifice of the zygomatico-facial canal, *fl* is the lachrymal fossa. (From Fuchs' *Textbook of Ophthalmology*.)

protecting sheath of fascia to be found at the posterior orbital foramina which encircles the nerves and vessels, passing through. In the mesial wall is to be found a couple of small foramina for transmitting the ethmoid nerves and vessels. A groove or foramen is to be found in the superior arch for the transmission of the supraorbital nerve and vessel. On the floor of the orbit is a groove for the infraorbital nerve and vessel. These pass out of the orbit through the infraorbital foramen, which is about 5 to 10 mm. below the center of the lower

orbital rim. Mesially is located the lachrymal sac and nasolachrymal ducts. Just inside the border of the superior arch to the nasal side is to be found a small spine, the origin of the trochlea. (Figs. 7, 8, 9, 10, and 11.)

Angles of the Orbit.—The external or anterior boundary of the orbit is, for the purpose of measurement and convenience in description, divided into four parts with corresponding angles.

These angles consist of two superior, known as external and internal superior, and two inferior, known as external and internal inferior angles. These are not always definite angles. In fact, in certain types of head, the angles are practically effaced in all except the inferior angles. The upper border of the orbit formed from the

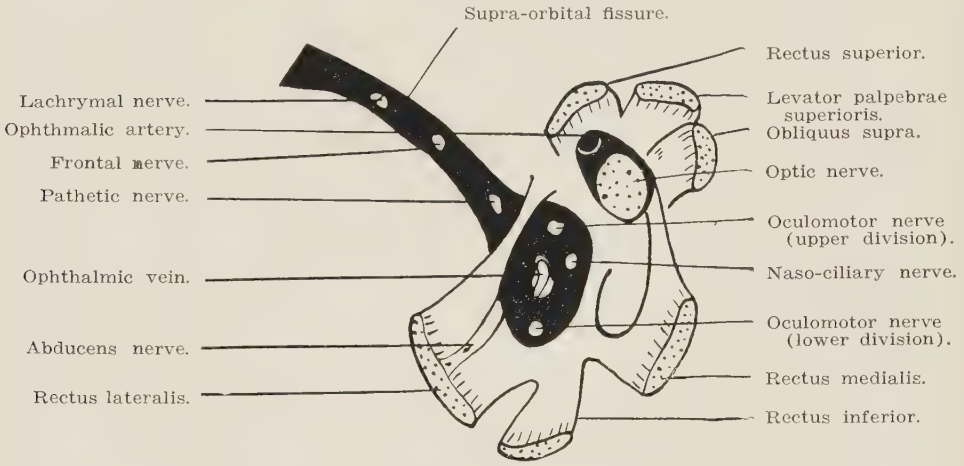

Fig. 7.—Apex of the orbit showing origin of the ocular muscles and their relations to the nerves and blood vessels. (After Poirer.) (From Landolt, *Defective Ocular Movements and Their Diagnosis.*)

frontal bone projects considerably farther than the lower border, thus permitting a greater range of vision downward than upward. The lateral border is from 8 to 10 millimeters shorter than the medial border, thus giving a greater range of vision externally. The whole general form of the orbit is so directed as to permit a wide range of movement of the ocular globe. The internal border, which is the fixing point of the orbit from which most measurements are made, is formed by the orbital process of the frontal and nasal process of the superior maxillary bones. At the union of these two bones and the lachrymal bones is a point known as the **dacryon.** This particular point is the point of measurement. The distance between one dacryon and that of the other is known as the interorbital space. The orbital openings vary according to the type of the skull, from practically a round opening to that of a quadrilateral.

Measurements of the Orbit.—The measurements by different craniologists of the various diameters of the orbit have varied considerably, but this is accounted for because of their using different methods. The interorbital distance of the female as given by Emmert is 36 millimeters and for males 37 millimeters. The following measurements given by him for breadth and height, respectively, are: in infants, breadth, 34.5 millimeters and height 29.2 millimeters; in adults:

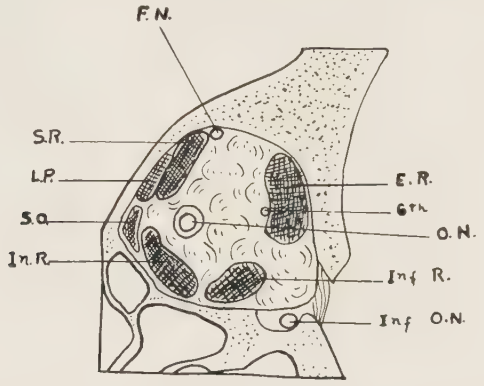

Fig. 8.—Section about 12 mm. behind globe. *S.R.*, superior rectus; *L.P.*, levator palpebrarum; *S.O.*, superior oblique; *In. R.*, internal rectus; *Inf. R.*, inferior rectus; *E. R.*, external rectus; *O.N.*, optic nerve; *F.N.*, frontal nerve; *Inf. O.N.*, infra-orbital nerve; *6th*, sixth nerve. (From Fuchs', *Textbook of Ophthalmology.*)

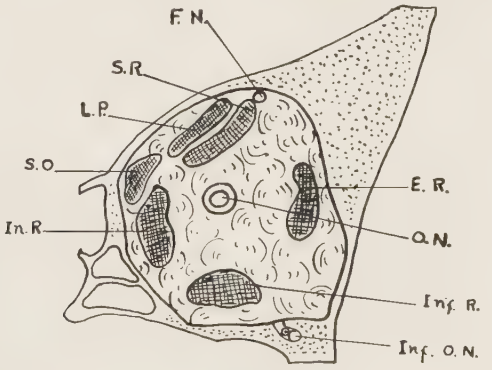

Fig. 9.—Section about 5 mm. behind globe. Letters as in preceding figure. (From Fuchs' *Textbook of Ophthalmology.*)

breadth, 39.8 millimeters, height, 33.16 millimeters for females; breadth, 41.6 millimeters, and height, 34 millimeters for males. Stevens, in a series of measurements, found but slight difference in these dimensions.

Contents of the Orbit.—The orbital cavity contains, besides the ocular globe, with its muscles, including the levator palpebrarum, a cushion of fatty substance and loosely connected tissue, which form

the bed in which the eye rotates, the blood vessels and nerves which supply the eye, the supraorbital vessels and nerves and the infra-orbital vessels and nerves, together with vessels of minor importance. It also contains the lachrymal gland, the caruncle, pulley of the tendon of the superior oblique, and the capsule of Tenon.

This fatty, fibrous cushion fills the entire apex of the orbit, except the space taken up by the muscles, nerves, and vessels, and thus fur-

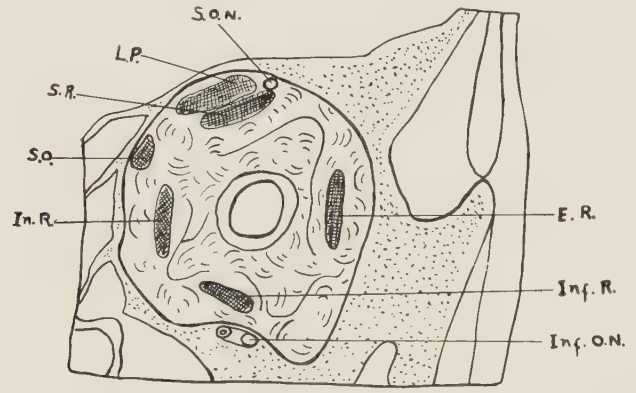

Fig. 10.—Section about 3 mm. in front of back of globe. Letters as before. *S.O.N.*, supra-orbital nerve. (From Fuchs' *Textbook of Ophthalmology.*)

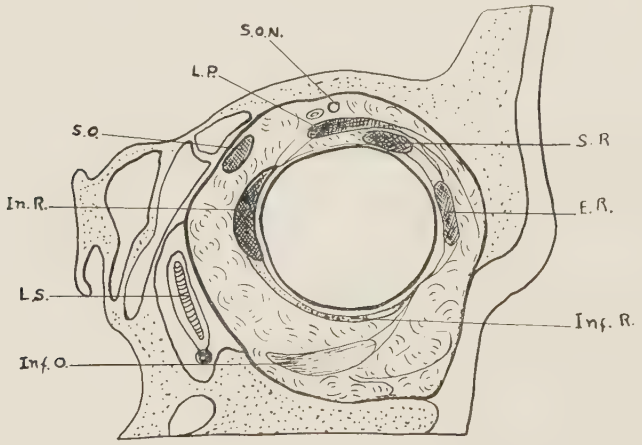

Fig. 11.—Section near equator of globe. Letters as before. *Inf. O.*, inferior oblique; *L.S.*, lachrymal sac. (From Fuchs', *Textbook of Ophthalmology.*)

nishes a fatty pad as a protection to the eyeball from injuries from in front. This fatty cushion is not of sufficient consistency to interfere with the free action of the muscles passing through it. While in a state of health, the fatty cushion supporting the eyeball usually remains about the same consistency and quantity, but in emaciating diseases and often in elderly subjects this fat is materially decreased

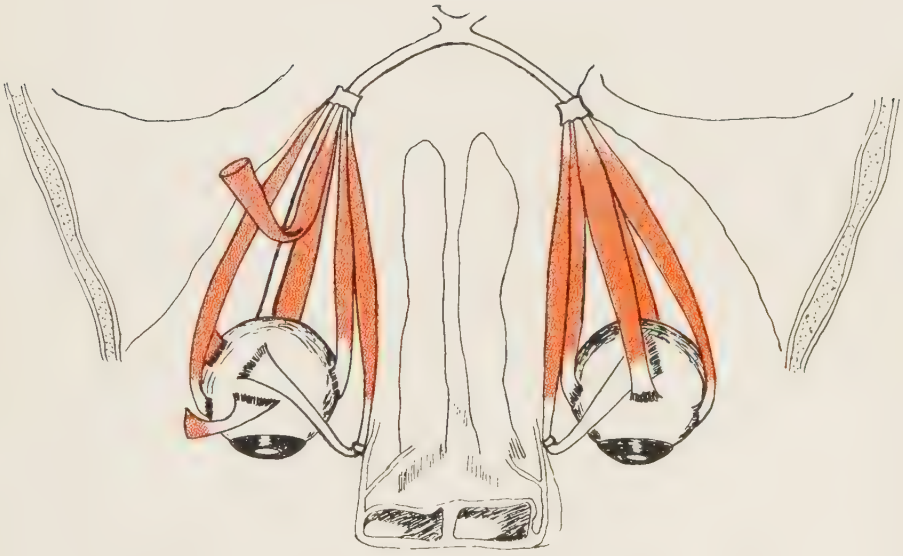

Fig. 12.—The motor muscles of the eyes. (From Stevens, *Motor Apparatus of the Eyes.*)

and causes considerable modification of the position of the eyeball. The character of this fatty cushion varies with its location, that immediately surrounding the capsule of Tenon, which envelops the eyeball, and also that surrounding the optic nerve consists of extremely fine fat cells, being held in loose meshes of connective tissue. The other part of the orbital fat is more firm and affords more support to the muscles.

Ocular Muscles

The ocular muscles are divided into external and internal, or extraocular and intraocular. The intraocular or internal muscles of the eye are the muscles of the iris and the muscles of the ciliary body, which will be discussed later.

We will first devote our attention to the external or extraocular muscles. Of these there are six in number, four straight and two oblique (see Fig. 7). The four straight muscles are the rectus medialis, or internal rectus, rectus lateralis, or external rectus, the rectus superior and rectus inferior. These four recti muscles take their origin from the apex of the orbit along the bony circumference of the foramen opticum, and from this point run forward, diverging as they go, forming a cone-shaped space known as the muscular funnel having its apex at the optic foramen, and its base at the eyeball (Fig. 12).

The optic nerve passes out from the optic foramen, running forward and outward through the axis of this muscular funnel to be inserted into the globe near its posterior pole. We shall now give a short anatomic description of each muscle.

Internal Rectus (Rectus Medialis).—This muscle has its origin in common with the other recti muscles in the inner surface of the optic foramen. The orbital periosteum at its apex forms a strong fibrous ring which surrounds the optic nerve as it enters the orbit. This ring is known as the annular ligament or tendon of Zinn. This fibrous band supplies an unyielding support for the origin of five of the ocular muscles.

The internal rectus has in addition a small fibrous attachment extending back to the sheath of the optic nerve. From its origin for about 8 or 10 mm. it is composed of a round tendon. At this point it speedily becomes muscular. These muscle fibers are straight parallel fibers and contain very little connective tissue, hence they tear easily in their longitudinal direction. The internal rectus passes forward from its origin almost parallel with the inner wall of the orbit and comes in contact with the eyeball near its equator and so continues until it reaches its insertion *about* 5.5 mm. to 6 mm. from the mesial edge of the cornea. The anterior end of the internal rectus is

a flat tendon about 1 mm. thick and 10 mm. wide and about 8.8 mm. in length. This muscle is 41 mm. in length, and weighs ¾ gram (Volkmann).

External Rectus (Rectus Lateralis).—This muscle has its origin from the tendon of Zinn and the fibrous sheath of the third nerve. It passes forward practically parallel with the outer orbital wall and comes in contact with the eyeball near its equator and follows it forward to be attached about 7 mm. from the lateral edge of the cornea. This muscle is second in strength, third in length, being 40.6 mm. long, and weighs $\frac{7}{10}$ gram. Its tendon is 4 to 5 mm. in length and about 9 mm. wide at its insertion, which, like the internal rectus, is practically straight and *vertical.*

Superior Rectus.—The superior rectus muscle has its origin at the tendon of Zinn and the border of the sphenoid fissure. From this point it passes upward and outward near the roof of the orbit and comes in contact with the eyeball near the equator and passes forward and is inserted into the sclera at about 7.7 mm. from the superior edge of the cornea near the median line. The superior rectus has a broad insertion, 10.6 mm. in breadth. This insertion is not usually in a straight line. The inner edge is in advance of its outer edge, and, according to Dwight[8] (Norris and Oliver, p. 97), most of its insertion is on the outer half of the eye. This is the weakest and longest of the recti muscles. Its length is 41.8 mm., while its weight is only ½ gram. The capsular sheath of the superior rectus is closely united to the sheath of the levator palpebrarum.

Inferior Rectus.—This muscle has its origin at the tendon of Zinn in common with the other recti muscles and passes along the lower border of the orbit underneath the eyeball and is inserted into the sclera about 6.5 mm. from the lower surface of the cornea. Its tendon is 5.5 mm. long, 9.8 mm. in width, and its insertion slightly curved forward with its center slightly to the nasal side of the horizontal meridian and its outer edge slightly in advance of the inner. This muscle is 40 mm. long and weighs ⅔ gram.

Superior Oblique.—This muscle has its origin at the tendon of Zinn, between that of the internal and superior recti from which point it passes upward and forward along the superior internal angle of the orbit. On approaching the orbital angle it is transformed into a round tendon which passes through a pulley, the trochlea. This is a fibrous ring-like opening situated at the orbital surface of the frontal bone, just beneath the inner angle of the brow. It is lined with a synovial membrane and permits free gliding movement of the round tendon of the superior oblique. In passing through the trochlea the tendon is bent backward and outward at about a 50° angle and passes

underneath the superior rectus and is attached to the sclera posterior to the equator and mostly outside the median line. Stevens[9] says:

"Dividing the ball by the equator and by a vertical median meridian, a small portion of the tendinous insertion is found in hypermetropic and in certain emmetropic eyes, at the upper posterior and inner quadrant as above remarked, while much the more extensive part of the insertion is in the upper, posterior, and outer quadrant of the sclerotic surface."

Fig. 13.—The relative insertions of the different muscles into the sclera (according to the data supplied by Professor Fuchs.) *S.O.*, superior oblique; *S.R.*, superior rectus; *I.O.*, inferior oblique; *Ex. R.*, external rectus. (Stevens, *Motor Apparatus of the Eyes.*)

Inferior Oblique.—This muscle differs from all the other ocular muscles in that it does not arise from the apex of the orbit. It has its origin from a slight depression on the orbital plate of the superior maxillary bone, at the inferior inner angle of the orbit and about 8 to 10 mm. from its border. From here it passes backward and outward, passing between the orbital wall and the inferior rectus, then between the eyeball and the external rectus, and is united to the sclera by a broad tendon at the superior external and posterior quadrant of the eye. The distance of this insertion from the optic nerve is from 5 to 7 mm. The breadth of its insertion is 9 to 10 mm.

Insertion of the Tendons.—It will be observed from the above descriptions that the insertions of the tendons of the various ocular muscles vary as to extent, position, and angle, and the measurements as given are only to be taken as averages. Fig. 13 taken from Fuchs, drawn by Stevens, shows the different variations observed by that master.

The four recti muscles have their insertion into the eyeball at different marginal distances from the cornea and are not usually perfectly concentric, nor are they perfectly symmetrical in their relation to the horizontal and vertical meridians. These variations are well shown in Fig. 14 taken from Hansell and Reber, p. 8.

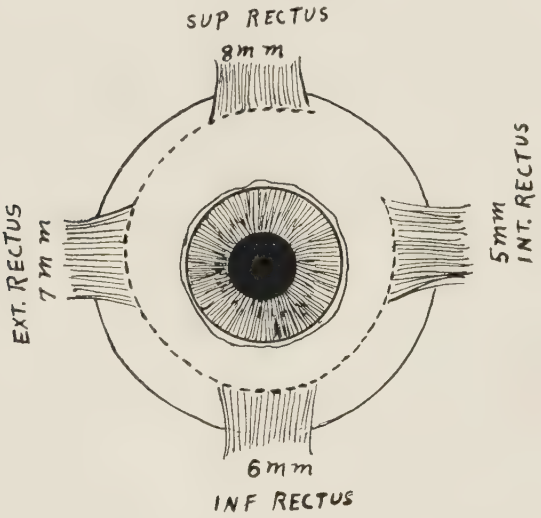

Fig. 14.—The spiral of insertions. (From Hansell and Reber, *Ocular Muscles.*)

The Ciliary Muscles.—There are two muscles in the ciliary body. The first muscle was discovered by Brucke, and about the same time by Bowman, and is usually called Bowman's muscle. The second muscle in the ciliary body was discovered by H. Muller, and is known by his name.

The Brucke-Bowman muscle was originally called the *tensor choroideae,* as it was supposed to make the choroid more tense when it acted (Fig. 15).

This muscle is the larger of the two and lies on the external surface of the ciliary body and runs from a point near Schlem's canal back toward the choroid, hence its name, *tensor choroideae.* The second muscle is located much deeper in the ciliary body, and its fibers run in a circular course.

With reference to the muscles of the ciliary body, Fuchs[10] has this to say:

"The ciliary muscle is composed of smooth muscular fibers, which do not present a compact mass, but are disposed in flat bundles, which are separated by connective tissue, and which interlace repeatedly so as to form a sort of plexus. For this reason, there is no well-marked separation between the two portions of the ciliary muscle; on the contrary, the longitudinal fibers by a very gradual transformation become bent so as to take a circular direction. Those bundles which effect the transition from fibers of one direction to those of another have been denoted by the name of radial bundles."

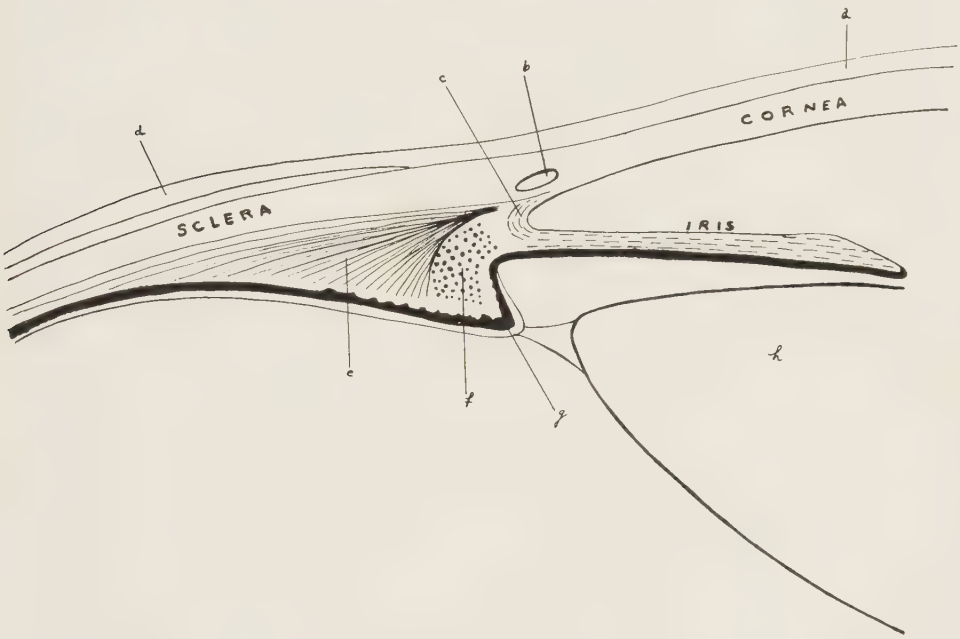

Fig. 15.—The circular and radial ciliary muscles. *a*, corneal conjunctiva; *b*, scleral sinus; *c*, pectinate ligament; *d*, scleral conjunctiva; *e*, radial ciliary muscle; *f*, circular muscle; *g*, ciliary process; *h*, lens. (From Baker in Norris & Oliver.)

Muscles of the Iris.—There are but two muscles in the iris. They are the *sphincter iridis* and the *dilator pupillae*. The sphincter iridis is about one millimeter broad, a flat, smooth muscle which lies in the iris stroma at the pupillary margin. The dilator pupillae is also situated in the iris stroma, and extends in a radial direction from the ciliary margin of the iris to the pupillary margin.

Arterial Supply to Ocular Muscles

The ophthalmic artery, a branch of the internal carotid, which enters the orbit through the orbital foramen at the external inferior side of the optic nerve furnishes the principal blood supply to the

ocular muscles. This vessel passes from the foramen forward and upward along the superior inner wall of the orbit, lying between the external rectus and the superior oblique. At the border of the orbit it divides into two main branches, the nasal and the frontal.

The ophthalmic artery in its course from the foramen forward passes over the optic nerve from its outer to its inner sides, and along this course it gives off several arteries which go to supply the lachrymal gland, the interior of the eye, the tissues of the orbit, and the six extraocular muscles.

The infraorbital artery, a branch of the internal maxillary, which is a branch of the external carotid, gives off an orbital branch, which supplies the orbital tissues and particularly the internal rectus, inferior oblique, and the lachrymal gland. This vessel supplies important collateral circulation in event of a destruction of the internal carotid.

Veins

There are two main veins which drain the orbit, the two ophthalmic veins, the superior and inferior, which unite behind the eyeball and pass backward between the heads of the external rectus, passing through the sphenoidal fissure, and enter the cavernous sinus. The superior ophthalmic is the longer of the two and corresponds closely with the ophthalmic artery (Fig. 16).

The inferior ophthalmic vein originates in the inferior part of the orbit, being formed from the vessels from the nose, face, the lids, and lachrymal passage, and passes underneath the eye to unite with the superior ophthalmic. One significant fact is to be noted with reference to the ophthalmic veins: they are devoid of valves and thus permit more readily serious infection of the deeper structures, and at times cavernous sinus thrombosis.

Nerve Supply to the Ocular Muscles

The ocular muscles receive their nerve supply from three cranial nerves. These are the third (Oculomotor, Oculomotorius, Motor Oculi Communis), the fourth (Trochlear), and sixth (Abducens). (Fig. 17.)

The Third, or Oculomotor, Nerve.—This nerve, the most important nerve supply to the eye, arises from a nucleus on the ventral floor of the third ventricle and aqueduct of Sylvius. Stevens[11] says:

"From this group of nuclear cells the fibers in various small groups pass forward through the nucleus ruber-tegmente to the pedunculus corporis mammillaris, then, before uniting, emerge from the brain substance at the line of the oculomotor groove between the two crura cerebri, and form a fasciculus of about a dozen cords which unite at

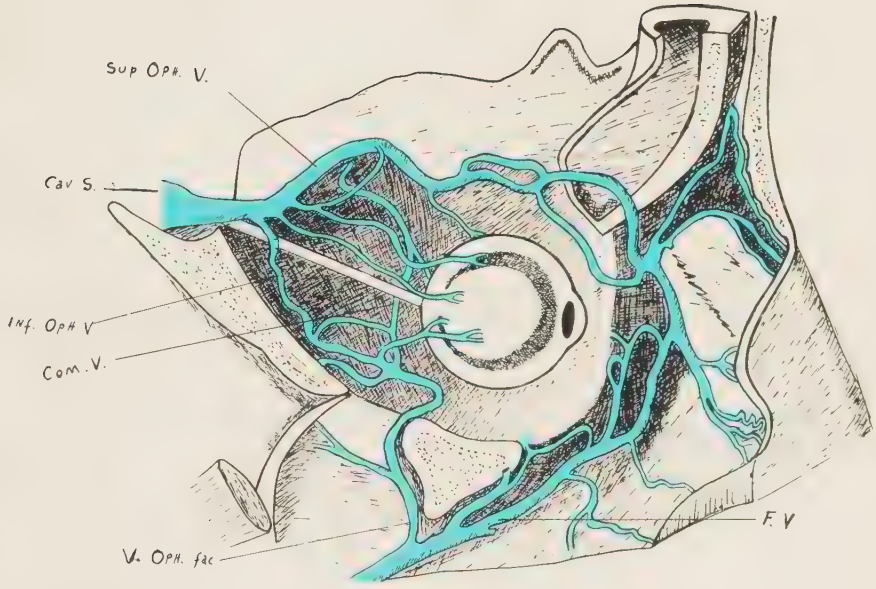

Fig. 16.—Veins of the orbit (from Sesmann). *Cav. S.*, cavernous sinus; *Com. v.*, communicating branch between *Sup. Oph. V.*, superior ophthalmic vein, and *Inf. Oph. V.*, inferior ophthalmic vein; *F. V.*, facial vein; *V. Oph. fac.*, ophthalmofacial vein passing through sphenomaxillary fissure. (From Dwight in Norris & Oliver.)

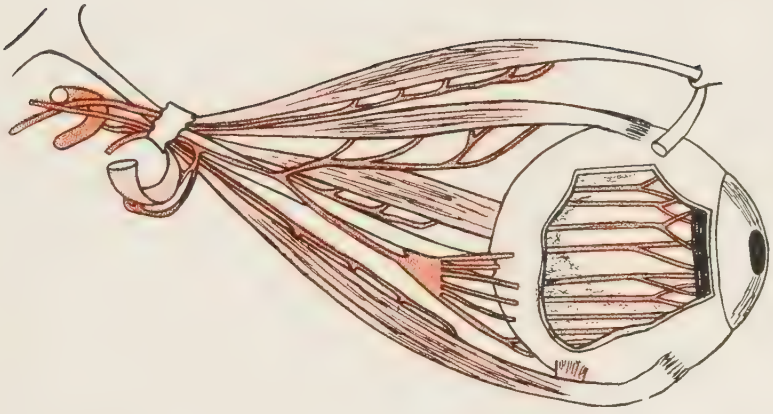

Fig. 17.—Distribution of the nerves of the muscles of the eyes. (From Stevens, *Motor Apparatus of the Eyes.*)

a short distance from their exit from the brain as the third cranial nerve. The nerve at its thickest part is about 3 mm. in diameter, and, according to Krause, contains about 15,000 fibers."

Fibers of most motor nerves are connected with both hemispheres. This has been questioned by some anatomists, but is now generally conceded. Alexander Bruce[12] claims that the anterior or ventral group of cells of the nucleus have a well-developed system of fibers, which connects each nucleus with its fellow of the opposite side. Duval and Labords (Stevens) have demonstrated a connection between the oculomotor nerve of one side with the abducens of the other.

The oculomotor nerve passes forward and outward between the posterior and superior cerebral arteries, emerges from the dura mater at a point where the tentorium cerebelli unites with the base of the skull, and from thence makes its way along the external wall of the cavernous sinus. At this point it receives anastomotic branches from the fifth and great sympathetic nerves. It passes through the sphenoidal fissure at its inner end. On entering the orbit the nerve is divided into two branches, the superior and inferior, and passes between the two heads of the external rectus muscle. The upper branch, the smaller of the two, runs forward and upward and innervates the superior rectus and the superior palpebrarum. The inferior branch supplies branches to the internal, and inferior rectus and to the inferior oblique. The oculomotor nerves enter the muscles which they supply from the orbital side of the muscle, with the exception of the inferior oblique, which it enters on its outer side.

Trochlear Nerve, or Fourth Nerve.—This nerve has its origin in a mass of gray cells in a plane of the front of the quadrigeminal body. The group of cells, known as the anterior trochlear nucleus, constitutes the principal origin of the fourth or trochlear nerve. These lie just behind the nucleus of the third nerve, from which there is no distinct boundary. Farther back and on the dorsal side of the posterior longitudinal bundle is another group of small cells which is known as the posterior trochlear nucleus.

Passing obliquely downward and backward from these nuclei these fibers unite into bundles and find their exit at the velum medullae anterius. At the roof of the aqueduct the fibers cross with their fellows of the opposite side. From this apparent origin, at the velum medullae anterius, at the point of contact of the two crura just behind the corpora quadrigemina, the nerve proceeds to the trochlear muscle as follows:[13]

"Turning outward, around the superior peduncle of the cerebellum, and forward across the crus cerebri, it reaches the anterior portion of the crus and proceeds along the transverse fissure and between the posterior cerebral and the superior cerebral arteries, and over the internal carotid arteries. Penetrating the dura mater beneath the ten-

torium, it continues its course along the outer wall of the cavernous sinus, in contact with and above the ophthalmic nerve and outside the abducens, obliquely crossing the third nerve, and enters the orbit by way of the inner end of the sphenoidal fissure. It is separated from the other contents of the cavernous sinus by a thin bony partition. As it enters the orbit, it passes above the external rectus over the levator palpebrae and superior rectus, entering the upper surface of the trochlearis muscle."

The trochlear nerve communicates with the sympathetic nerve in its course through the cavernous sinus. It is also said to be connected with the ophthalmic branch of the fifth nerve, as is found with the

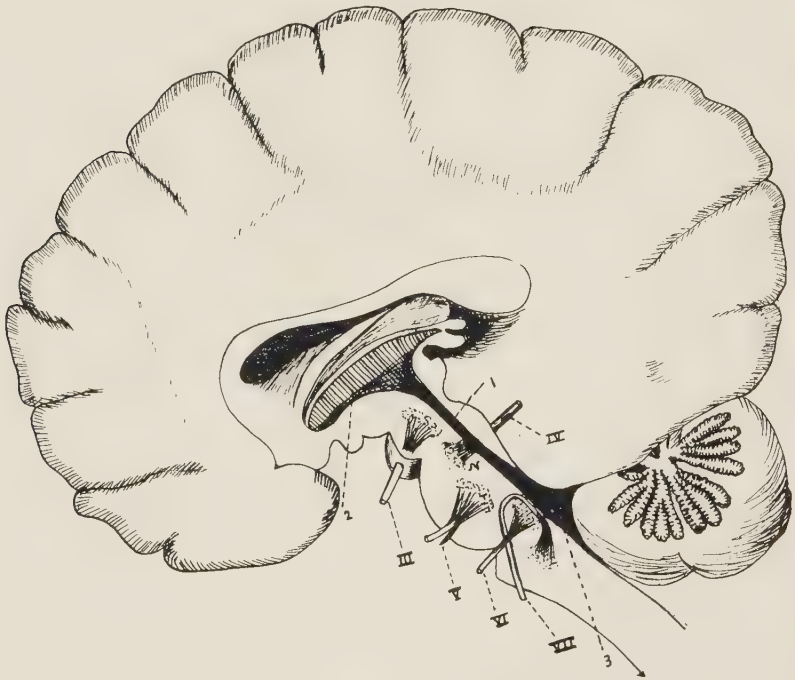

Fig. 18.—Diagrammatic representation of midbrain with approximate situations of the nuclear groups from which arise the third, fourth, fifth and sixth nerves. (From Stevens, *Motor Apparatus of the Eyes.*)

third nerve, and small filaments connect it with both the carotid plexus, and to the infratrochlear nerve.

The Abducens, or Sixth Nerve.—This nerve arises from a nucleus at the floor of the fourth ventricle just posterior to the nucleus of the motor root of the fifth nerve, in proximity with the seventh nerve nucleus. From this nucleus the nerve passes forward toward the medial line, and makes its exit from the medulla just behind the pons. A small bundle of fibers unites the nucleus with the abducens nerve of the opposite side. At a point between the anterior pyramid and the pons, the nerve has its apparent origin. From here it passes directly for-

Fig. 19.—Aileron of the superior rectus muscle seen in profile from the internal side. (From Motais, *Anatomie de l'Appareil Moteur de l'Oeil.*)

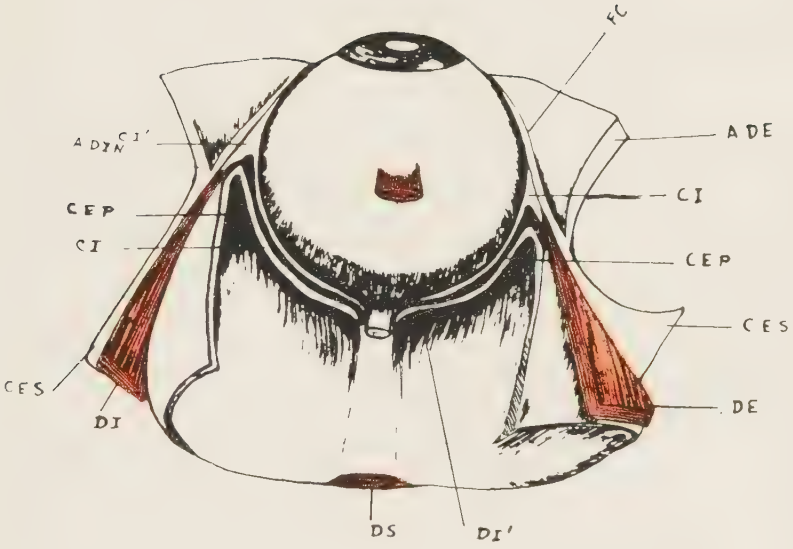

Fig. 20.—Capsule of Tenon. Connections of the internal capsule with the anterior extremity of the muscle and the tendon. *CI*, internal capsule surrounding the anterior extremity of the external rectus *DE*, from the point where the external ligamentous aileron *ADE* and the profound portion of the sheath *CEP* leave the muscle *CI'*, internal capsule connecting in like manner with the anterior extremity and the tendon of the internal rectus *DI*, its ligamentous aileron *ADIN* and the profound portion of the sheath *CEP*. *CES*, *CES*, superficial portion of the sheath of the internal and external rectus muscles. *FC*, subconjunctival fascia. *DS*, superior rectus muscle. *DI'*, inferior rectus muscle. (From Motais, *Anatomie de l'Appareil Moteur de l'Oeil.*)

ward, piercing the dura mater, enters the cavernous sinus, in which it lies close to the floor and in contact with the lateral side of the internal carotid artery. The abducens enters the orbit through the sphenoidal fissure beneath the other nerves and passes between the two heads of the external rectus. This nerve also has connections with the sympathetic plexus and the ophthalmic nerve.

Nuclear Origin of the Ocular Nerves.—The nuclear origin of the three nerves which supply the ocular muscles is to be found in the midbrain on the floor of the third ventricle, extending from the level of the posterior commissure to in front of the anterior corpora quadrigemina and backward to about half-way between the anterior and posterior quadrigemina. These consist of groups of cells, which lie in more or less order from before backward, extending along the floor of the aqueduct of Sylvius to the fourth ventricle. Each group of cells constitutes the nuclear origin of one of the motor nerves of the eye, consisting of the third, fourth, sixth, and also nuclear origin of the fifth nerve.

Fig. 18, taken from Stevens (p. 75), will aid the student in getting a better mental grasp of the nuclear origin of the various nerves supplying the ocular muscles.

Capsule of Tenon

The capsule of Tenon is a fibrous tissue membrane which invests the eyeball and muscles and attaches it to the orbital walls and forms protection and support to both. By means of this system of fascia, the contents of the orbit are fixed in place. It is owing to them that the eye does not lose its place when performing its movements, but turns about a fixed center. These fasciae pass from the muscles to the walls of the orbit and act as restraining apparatus which prevents the extreme excursion of the eyeball. Galen first mentions this membrane as *Tunica Sexta*. After him, R. Columbus in the sixteenth century described the capsule under the name of *Tunica Innominata*, but these descriptions were vague and no definite study was made of this membrane until it was described by Tenon[14] in 1806. His description was so accurate and definite that this membrane or capsule has been known as Tenon's capsule since his writing. After Tenon, Malgaigne, Baudrue, Bonnet, and others made interesting investigations of this capsule, but we owe to Motais' painstaking work much of our present knowledge of this capsule.[3] (See Figs. 19 and 20.)

The difficulty of presenting an accurate description of this extensive, complicated membrane can be appreciated from the following remarks of Dwight[15] with reference to it. He says:

"The complications of this membrane are limited only by the perverted ingenuity of those who describe it."

This membrane may best be described by dividing it into three parts:

1. Internal capsule.
2. External capsule.
3. Orbital ligaments.

Internal Capsule (Tenon's Capsule, Orbital Fasciae).—This capsule forms the real socket in which the eye is moved. The capsule begins at the fibrous ring about the optic nerve as it makes its entrance into the orbit and covers the optic nerve as an outer sheath and follows it to the sclera, to which it is closely attached about the scleral entrance of the optic nerve. This capsule lines the orbital fat about the surface of the eyeball and on its bulbar surface is covered with epithelial layer. Between this membrane and the eyeball are to be found fibrous thread-like attachments. The space between the eye and this capsule is supposed to be a lymph space, connected with the supravaginal space on the outer side of the dural sheath.

Behind the insertion of the muscles the capsule is reflected backward and forms a common aponeurosis which connects the ocular muscles. This portion of the capsule is known as the muscle capsule. It here invests each muscle, being thickest at its tendon and extending backward on each until it is lost in the muscle sheath.

External Capsule.—This portion of Tenon's capsule begins where the tendons are supposed to perforate the capsule. As a matter of fact, they do not perforate the capsule, but the capsule is merely reflected back upon the muscle. This capsule may be considered as two layers of fibrous tissue, one on the inner and the other on the outer surface of the muscle at its tendinous insertion. The inner layer is reflected back upon the muscle several millimeters behind its insertion. The external layer is closely attached to the tendon until it arrives within a millimeter or two of its insertion, and then it passes forward and is attached to the sclera near the cornea. The capsule in this location surrounds the anterior portion of the eye as a fibrous band. (Figs. 21 and 22.)

Orbital Ligaments. (Ligamentous Ailerons.)

Connecting with the above-described capsule at the tendons of the internal and external muscles are strong fibrous bands, known as check ligaments, which run from these tendons and are attached to the bony orbits internally and externally. These fibrous bands at times contain muscle fibers and are sometimes called orbital muscles.[16]

Fuchs gives two ligamentous connections with the superior oblique. The superior oblique has two check ligaments, according to Duane, one attached to the muscles before it reaches the trochlea, and a second attached to the tendon between the trochlea and the eyeball.

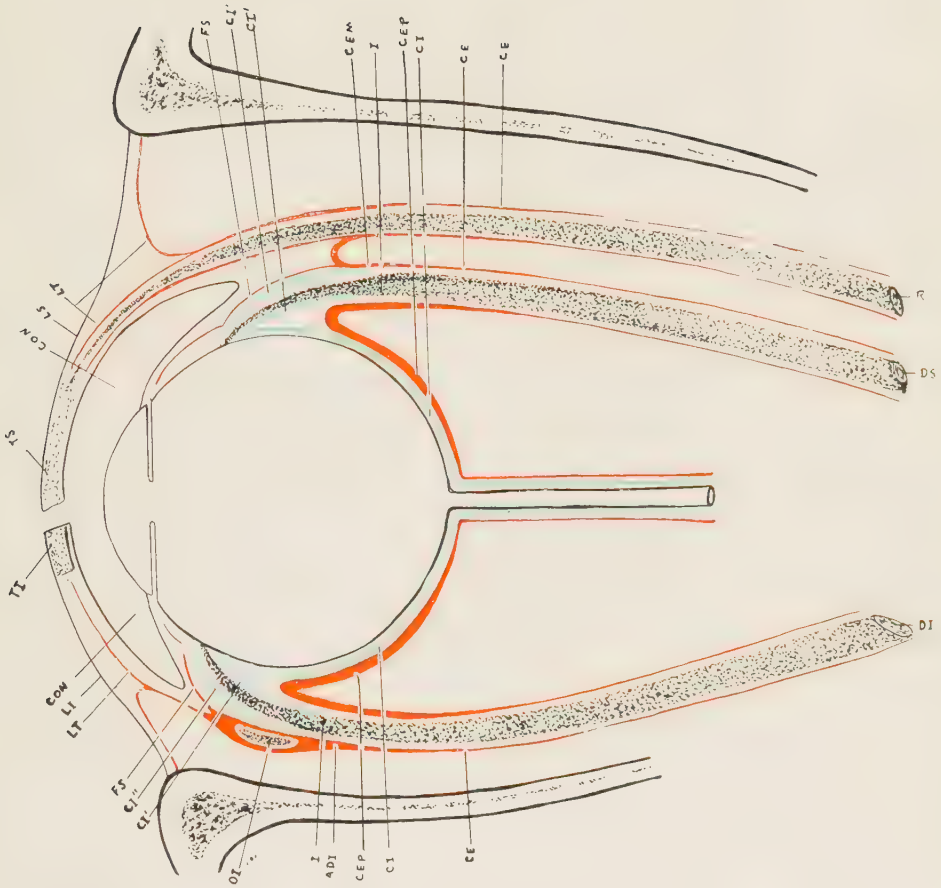

Fig. 21.—Diagram (schematic) of the capsule of Tenon in man. Vertical section passing through the superior and inferior rectus muscles.

CE, CE, CE (red), aponeurosis or external capsule forming the sheath of the muscles. *CEP, CEP,* profound portion of the half of the globe. *CEM,* superficial portion of the sheath of the superior rectus muscle folding back to form the sheath of the muscle lifting the eyelid. *LT, LT,* terminal lamellae of the aponeurosis returning to the orbit and to the tarsal cartilages. *FS, FS,* subconjunctival fascia.

CI, CI (blue), bulbar or internal capsule, serous membrane of the eye. *CI',* internal capsule folding back under the profound side of the tendon and the muscle, which it covers up to the point I, where the external capsule leaves the muscle. *CI''*, internal capsule in front of the tendon and the anterior extremity of the muscle circumscribing a serous sac indicated by the blue stippling. It stops behind the point where the external capsule leaves the muscle to proceed toward the orbit; forward, it is inserted into the sclerotic with the tendon, instead of extending up to the edge of the cornea like the subconjunctival fascia, *FS.*

ADI, aileron of the inferior rectus *DI* redoubling upon itself to envelope the little oblique muscle, *OI. DS,* superior rectus muscle. *R,* lifting muscle of the eyelid. *LS,* superior tarso-orbital ligament. *LI,* inferior tarso-orbital ligament. *TS,* superior tarsal cartilage. *TI,* inferior tarsal cartilage. *CON, CON,* conjunctival space. (From Motais, *Anatomie de l'Appareil Moteur de l'Oeil.*)

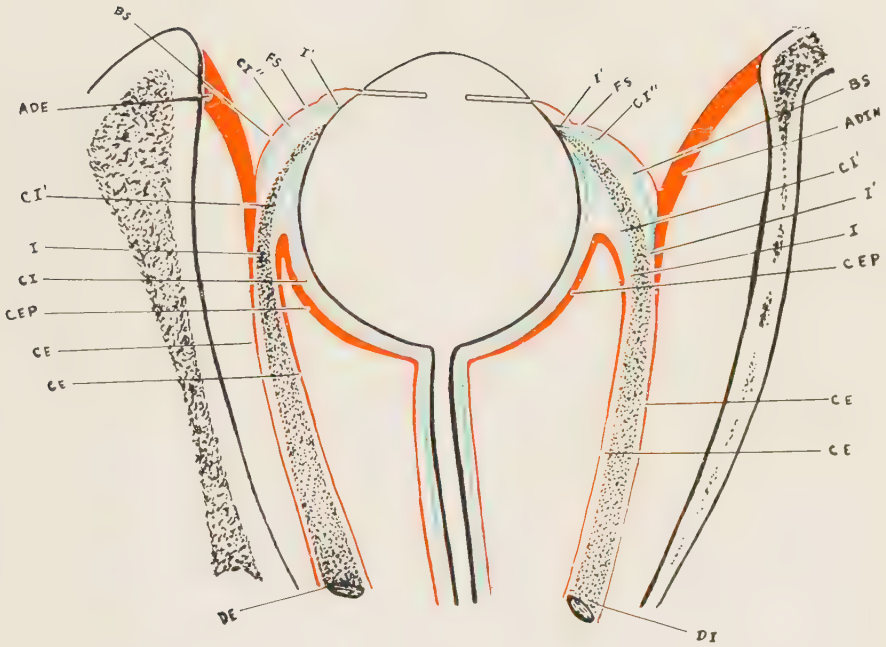

Fig. 22.—Diagram of the capsule of Tenon in man. Horizontal section passing through the external and internal rectus muscles.

CE, CE, CE, CE (red), aponeurosis or external capsule forming the sheath of the muscles. *CEP, CEP*, profound portion of the sheath of the muscles folding backward to cover the posterior half. *ADE*, external ligamentous aileron. *ADIN*, internal ligamentous aileron. *FS, FS*, subconjunctival fascia.

CI (blue), internal capsule, *CI'* internal capsule folding back under the tendon and the muscle up to the point *I*. *CI''*, *CI''*, internal capsule in front of the tendon and the muscle which it covers up to the point I, where the aileron leaves it; circumscribing a serous sac *BS, BS*, indicated by the blue stippling; then stopping before the tendinous insertion *I'*.

DI, internal rectus muscle. *DE*, external rectus muscle. (From Molais, *Anatomie de l'Appareil Moteur de l'Oeil.*)

Motais found similar bands extending from the recti muscles and the superior oblique; but these are much smaller than the internal and external. There are also similar bands connecting the superior palpebrarum and thus the upper lid. There are also bands connecting the superior rectus and inferior oblique muscles, and a rather strong ligament connecting the inferior rectus with the orbit. These check ligaments are very important from a surgical standpoint in operating upon cases of squint.

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[3]Motais: Anatomie de l'Appareil Moteur de l'Oeil, Paris, 1887.
[4]Dwight: Norris and Oliver, System of Diseases of the Eye, Philadelphia, Lippincott, 1898, i.
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CHAPTER IV

PHYSIOLOGY OF THE MUSCLES OF THE EYE

In order to understand the actions of the ocular muscles, both individually and collectively, it is necessary to know not only their origin and insertions but also their relative strength and their peculiar individual plane of insertion to the eyeball.

The eyeball is situated in Tenon's capsule, and moves about in it by means of the action of the various ocular muscles similar to the movements of a round ball in a socket.

For purposes of study, the head is divided into different planes and the orbit into others, while the eye itself has its axes, poles, meridians, and equator. We shall discuss these elementarily before taking up the actions of the muscles.

Planes of the Head.—The two important planes of the head are the *median* and the *horizontal*. The *median* plane is a fixed vertical plane which divides the head into two hemispheres. It is a vertical plane passing from above downward between the hemispheres of the cerebrum and midway between the orbits, thus dividing the head into two equal parts, as indicated by the line *ab* in Fig. 23.

The *horizontal* or transverse plane in ideally constructed heads, for purposes of study, should pass through the center of rotation of each eye, through the chiasm, and would pass backward approximately between the cerebrum and cerebellum, and remain at all times perpendicular to the median plane.

Frontal Planes.—Frontal planes are vertical planes at right angles to median planes.

Sagittal Planes.—The sagittal plane is a vertical plane running from before backward, and is either in or parallel to the median plane.

The two parts separated by a median plane are the right and left hemispheres. The two parts separated by a horizontal plane are the superior and inferior. The two parts separated by a frontal plane are the anterior and posterior.

Point, Plane, and Line of Regard.—The point of regard, or the point of fixation, is the point fixed by the two eyes, being a point where the visual lines of the two eyes meet in binocular vision.

The Line of Regard, or visual axis, is a straight line drawn from the object fixed through the center of rotation of the eye to the macula.

The Plane of Regard is a plane drawn by and included within the two lines of regard.

The Field of Regard is the field which the point of regard may traverse. This is always less than the visual field.

The Base Line is a line drawn between the center of motion of the two eyes.

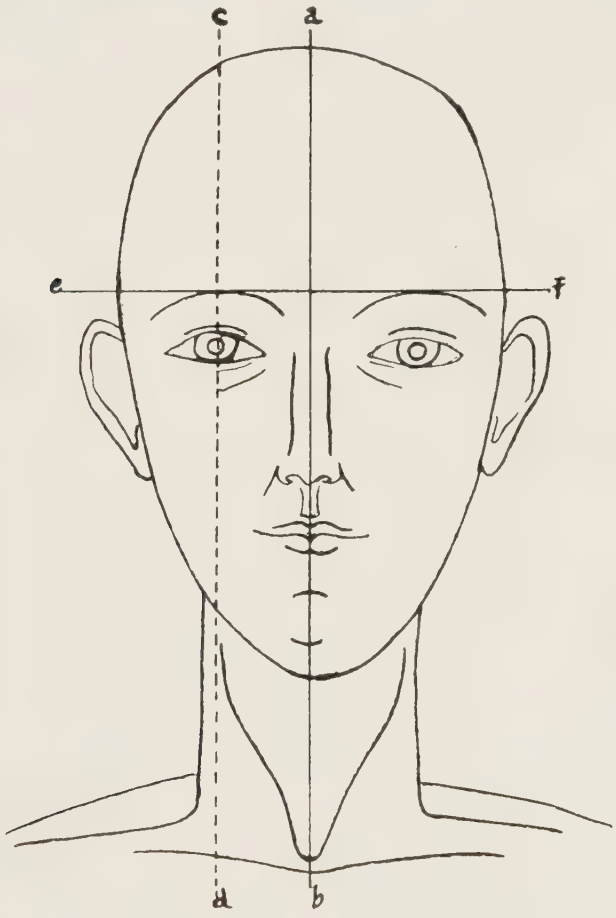

Fig. 23.—Planes of the head. (From Stevens, *Motor Apparatus of the Eyes.*)

The median line of the plane of regard is a line drawn from the middle of the base line to the object of fixation.

There are two *planes of reference* which are of particular value in the study of the ocular movements. As above stated, these are the *median* and *horizontal* planes. Fig. 24 taken from Savage[1] represents these planes, and is aptly described by him as follows:

"Figure 1 [Fig. 24] represents these two planes, also the two eyes in ideal orbits. The line *AB* is the fixed vertical median plane of the

head; *CD* is the fixed horizontal plane of the head; *gh* and *gh* are the vertical axes of the eyes, and *ef* and *ef* are the horizontal or transverse axes of the eyes. In orbits, whether well formed or malformed, the eyes must have their vertical axes, *gh*, parallel with *AB*. In well-formed orbits, the eyes must have their transverse axes, *ef* and *ef*, lie wholly in the fixed horizontal plane of the head, *CD*. This is true whether the eyes are in the primary position or rotated in either one of the four cardinal directions or into any oblique position. Eyes contained in malformed orbits—malformed in the sense of being too

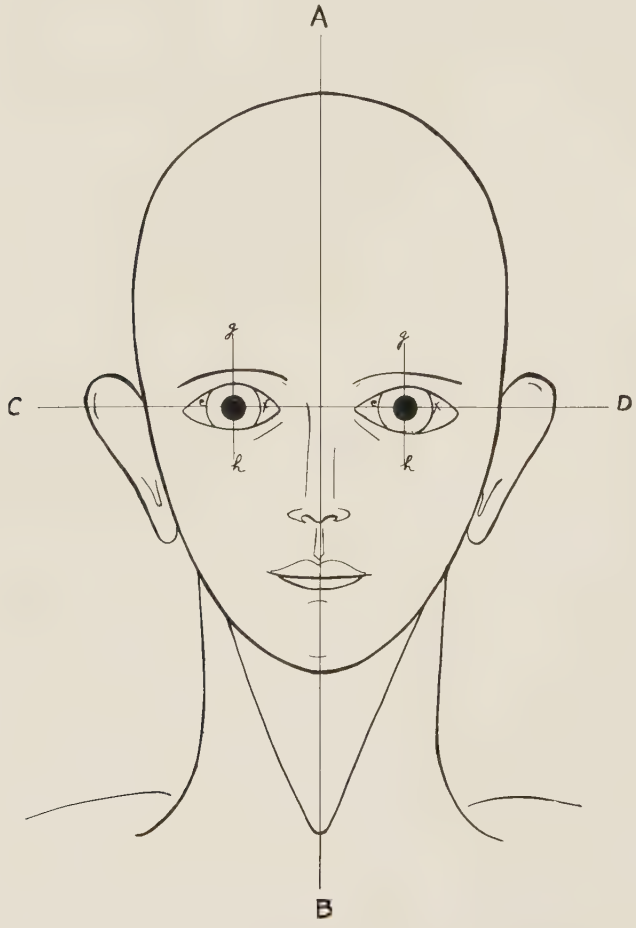

Fig. 24.—The horizontal and median planes of the head. (Modified from Savage, *Ophthalmic Myology.*)

low or too high—cannot have their transverse axes lie in the fixed horizontal plane of the head, but these axes must be parallel with this plane, whether above or below it, and that, too, regardless of the position occupied by the visual axes.

"The parallelism of the vertical axes of the eyes with the median plane of the head, and the including of the transverse axes of the eyes within the horizontal plane of the head, give us *correct* ideas of

verticality, horizontality, and obliquity of lines in space, and that, too, regardless of the position of the head. A line that is seen vertical, when the head is in the primary position, will appear vertical still if the head be inclined 45 degrees to the right or left, or to any other extent."

Poles of the Eye.—The *anterior pole* of the eye is the midpoint of the cornea in front. The *posterior pole* is the midpoint of the sclerotic behind (Maddox). (Fig. 25.)

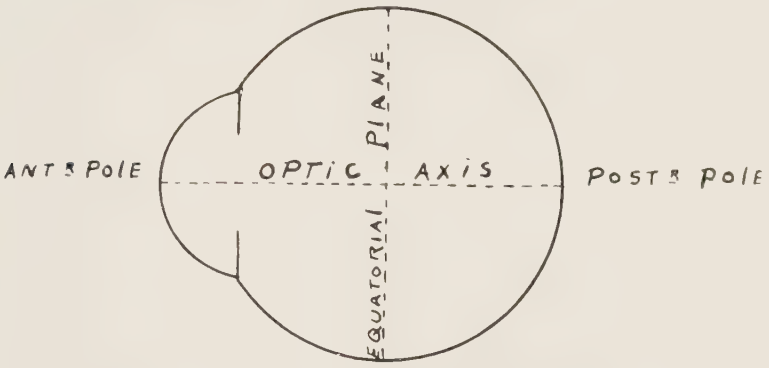

Fig. 25.—Poles of the eye. (From Maddox, *The Ocular Muscles.*)

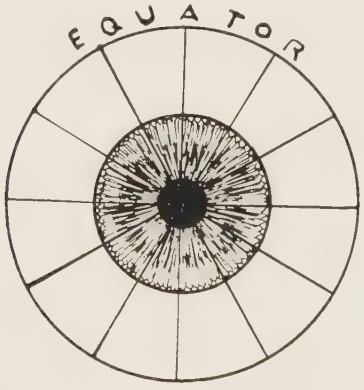

Fig. 26.—Equator of the eye. (From Maddox, *The Ocular Muscles.*)

Savage[2] claims that all corneo-retinal meridians cross at the macula, and for that reason the macula must be the posterior pole. Reasoning thus, he claims the optic axis and visual axis are identical.

The Optic Axis of the eye is a line running from the center of the cornea through the center of rotation to the retina, thus connecting the two poles.

The Visual Axis is a straight line drawn from the fixed object through the center of rotation to the macula.

The Equator of the eye is a belt of the globe midway between the two poles. (Fig. 26.)

Meridians are circles which pass through both poles with the optic axis of the eye as their common diameter.

In studying the eye, it is treated as though it were a sphere. This is not strictly true, as it is usually flatter before backward and with an extra curved surface in front, the cornea.

Center of Motion.—According to Helmholtz, the length of the normal eye is 23.266 mm., and according to Donders, the center of motion in the emmetropic eye is 13.45 mm. behind the anterior surface of the cornea. In the hypermetropic eye it is less, and in the myopic eye, more, depending entirely on the diameter of the eye. Thus it will be observed that the center of motion may be said to be 1.5 mm. posterior to the center of the globe. Most writers refer to the center of the globe as the center of motion. In a recent article in the *American Journal of Ophthalmology*, George, Torens, and Lowell[3] have presented a very interesting investigation made by them with reference to the center of motion. After examining several hundred eyes with a specially constructed instrument, they came to the conclusion that the center of motion is "15.4 mm. posterior to the corneal vertex and 1.65 mm. nasalward from the visual axis." This, you will observe, is about 3.5 mm. posterior to the center of the globe and about 2 mm. farther back than is given by other authors. This subject deserves our careful study, as it is a vital point in the correction of squint by accurate measurements.

Movements of a Sphere (Maddox).—A freely movable sphere is capable of six independent motions. It can be translated as a whole in three directions at right angles to each other. The axes about which a sphere may rotate are the *vertical* axis, the *transverse* or horizontal axis, and the *sagittal* or *fore* and *aft* axis. Rotation in any other direction becomes a rotation about two or more of these axes. A sphere which possesses all three degrees of rotation can rotate in any conceivable direction. We will now consider the ocular motions as related to those of a sphere.

Ocular Motions.—Since the eyes are fixed in the orbit, they cannot be translated in space in any direction. We have seen in the study of the anatomy of the orbit and its contents that the eye is freely movable in many directions. We shall now see about which axes the eye is able to rotate.

The eye is privileged to rotate only about its vertical and horizontal or transverse axes, and to a very limited extent about its fore and aft (sagittal) axis. These axes are considered fixed in the head; thus we see that the eye movements are practically limited to rota-

tions about the two planes, the vertical and the horizontal. The eye is still able to rotate about all conceivable diameters of one plane, *Listing's plane*, that plane in which the vertical and transverse axes lie. Maddox[4] gives us Listing's plane as follows:

"Listing's plane passes through the center of motion of the eyes, and is a vertical transverse plane (corresponding to coronal section) fixed in the head, and perpendicular to the fore-and-aft axis, about which rotation is denied."

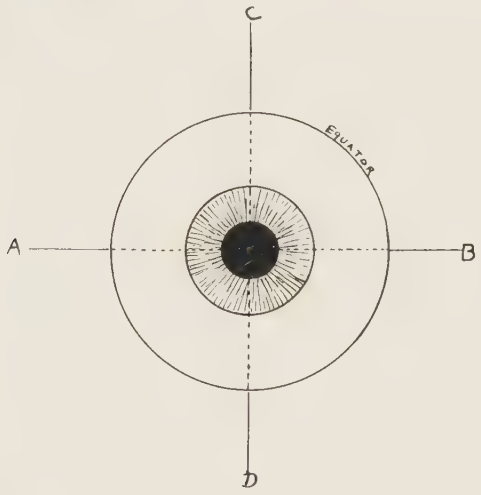

Fig. 27.—Horizontal and vertical axes. *AB*, horizontal axis; *CD*, vertical axis.

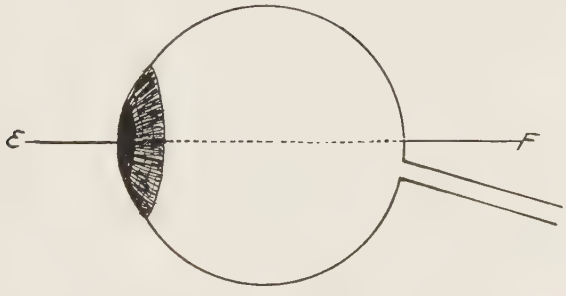

Fig. 28.—Visual axis. *EF*, fore-and-aft axis.

Primary Position.—When the head is held erect and the eyes are directed to infinity along the horizontal plane, they are said to be in their *primary position.*

Primary Axes.—We will observe that the movements of the eye can be said to turn about three primary axes, all of which are perpendicular to each other, and cross at the center of rotation. (Figs. 27 and 28.)

The vertical axis, about which all lateral (right and left) movements take place.

Horizontal or transverse axis (i.e., from right to left) about which all vertical (up and down) movements take place.

Sagittal axis (fore-and-aft) (Maddox) runs from before backward in line of vision, and all movements about this axis are wheel-like and are called torsions, by which the upper extremity of the eye is turned in or out (Duane).

Actions of the Ocular Muscles.—In studying the plane of action of any ocular muscle, it at once becomes necessary to determine the plane of that particular muscle.

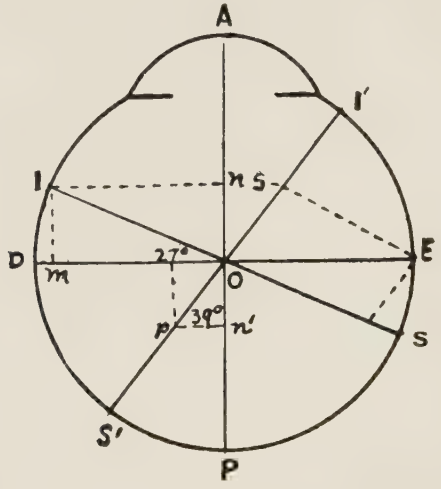

Fig. 29.—Horizontal section of a *right* eye. *A* is the anterior pole of the eyeball, and *P* is the posterior pole, so that *AP* is the optic axis. The line *DE* is the transverse axis; *IS* is the axis of rotation for the superior and inferior recti, and *I'S'* is the axis of rotation for the obliques. (From Maddox, *The Ocular Muscles.*)

Savage[5] says:

"The axis of rotation of the eye by any one muscle must be at right angles to the plane of rotation for this muscle. This plane must bisect the muscle at its origin and at its attachment, and must also pass through the center of rotation of the eye.

"There are three points through which this plane must pass, viz., the center of rotation of the eye, the center of the origin of the muscle, and the center of attachment of the muscle."

There are only two muscles of the eye whose actions correspond to any of the planes of the eye. These two are the rectus internus (medialis) and the rectus externus (lateralis). The planes of these two muscles are practically identical with the horizontal plane of the eye, and they may be considered to be the only muscles which rotate the eyes about one of the primary axes.

Rectus Internus.—Adducts the eye practically directly inward along the horizontal plane.

Rectus Externus.—Abducts the eye, causing it to turn outward along the horizontal plane. These two muscles may be said to be the only ones of six muscles to have a simple action, as the action of the four other muscles is complicated, and they do not move the eye in any one of the primary axes. Savage claims that often the actions of the internal and external recti are not simple, in that they either move the eye slightly above or below the horizontal plane due to faulty attachments to the globe.

Rectus Superior.—It will be observed from the anatomy of the muscles that this muscle runs from the apex of the orbit forward, upward, and outward, hence its axis does not correspond with either the sagittal (fore-and-aft) or median axes of the eye. Its insertion being in front of the center of rotation, it elevates the eye and adducts it, and at the same time it rolls (intorts) the upper meridian of the eye inward. It then is an elevator, adductor, and intorter. Fig. 29 from Maddox gives the planes of action of the superior and inferior recti and the two obliques.

The plane of the superior rectus makes an axis of about 30 degrees from the transverse (horizontal) axis (Mauthner).

Savage[5] says:

"For simplicity of study, it may be considered, therefore, that there is a common plane of rotation for both the superior and the inferior recti, and that the plane is vertical, forming an angle of 27 degrees with the vertical anteroposterior plane of the eye. The axis of rotation must be at right angles to the muscle plane, and consequently must form an angle of 27 degrees with the transverse axis, but in the horizontal plane with it."

Rectus Inferior.—The inferior rectus, as has been seen (see Anatomy), deviates outward from its origin on its way to its attachment to the eye anterior to the center of rotation. It causes the eye to turn down and inward and at the same time tilts the upper meridian of the cornea outward. Hence it is seen that the inferior rectus is a *sursumductor, adductor,* and *extorter.*

Oblique Superior.—The complicated anatomy of the superior oblique does not make the study of its actions more intricate, as its plane of action is to be taken from the trochlea to the muscle's insertion into the eyeball, the trochlea being from a physical standpoint the origin of the muscle. The muscle passes from the trochlea backward and downward and is inserted in the superior portion of the eye posterior to its center of rotation (the equator). Its chief action is to turn the vertical meridian of the eye inward, but since it is

attached to the posterior pole of the eye, it draws this up and causes the cornea to turn downward, and since it is attached beyond the center of rotation, it causes the cornea to face outward. Thus, the superior oblique is an *intorter, depressor,* and *abductor.*

Oblique Inferior.—This muscle has its origin in the margin of the orbit near its inner angle. It runs upward and backward to be inserted into the eye posterior to its equator. Being inserted in the posterior half of the eyeball, it acts to pull this portion of the eye down and in and thus turns the cornea up and out; at the same time it turns the vertical meridian outward (extorts). It is then an *elevator, abductor,* and *extorter.* The plane of action of the oblique muscles forms an angle of from 35 to 40 degrees with the visual axes (Stevens).

Main and Subsidiary Action.—Preeminent and subsidiary (Maddox); principal and subordinate (Savage).

Each ocular muscle, with the exceptions of the internal and external recti, has a main and a subsidiary action. The main action dominates the movement of the eye in the plane of the action of the individual muscle, and when the eye has turned out of the particular muscle plane its action becomes subsidiary, and some other muscle whose plane more nearly corresponds with the optic axis in that position becomes the main or dominating muscle. These actions are expressed by Duane[6] as follows:

"The main action increases as the subsidiary decreases, and vice versa.

"The main action increases in one special direction of the gaze. These facts are shown in the table subjoined.

"Superior Rectus: *Main action* moves eye up; action increases as eye is turned out; becomes nil when eye is turned in.

"*Subsidiary action* adducts eye and rotates vertical meridian inward; action increases as eye is turned in. Raises upper lid.

"Inferior Oblique: *Main action* moves eye up; action increases as eye is turned in; becomes nil when eye is turned out.

"*Subsidiary action* adducts eye and rotates vertical meridian outward; action increases as eye is turned out.

"Inferior Rectus: *Main action* moves eye down; action increases as eye is turned out; becomes nil when eye is turned in.

"*Subsidiary action* adducts eye and rotates vertical meridian out; action increases as eye is turned in. Pulls down lower lid.

"Superior Oblique: *Main action* moves eye down; action increases as eye is turned in; becomes nil when eye is turned out.

"*Subsidiary action* adducts eye and rotates vertical meridian in; action increases as eye is turned out.

Synergistic Action.—Muscles which aid the main acting muscle in turning the eye in a certain direction are called synergists.

Antagonistic Action.—Muscles which rotate the eye in opposite directions to the plane of a certain muscle are called antagonists. There

are only two perfect antagonists. These are the internal and external recti. The superior and inferior recti antagonize one another in their main action, but are synergistic in that they are adductors. The two obliques are antagonists in all actions, except that they both are abductors.

The four recti are antagonists to the obliques in that they draw the eye backward, whereas the oblique draws it forward (Duane).

Associates.—Every eye muscle has a true associate in the other eye. The right internal rectus with left external, and vice versa; the superior rectus of one eye with inferior oblique of the opposite; the inferior rectus with the opposite superior oblique.

The general control of the eye in all directions is well expressed by Dwight,[7] who says:

"Practically, it is to be remembered that it is very unlikely that one or even two of these muscles ever act alone. The accuracy of any movement is due not alone to the pull of the muscles to which it is usually ascribed, but also the graduated resistance of the antagonists. Probably the simplest movement of the eye is made by more or less active concurrence of all the muscles."

Along the same line, Browne and Stevenson[8] remark:

"The eyeball does not turn upon a pivot, nor is it seated in a well-fitting socket like the acetabulum; it is supported in a bag—the capsule of Tenon—and for every movement it is drawn upon by all the muscles. It is not turned simply by one muscle pulling and the opponent relaxing, as it would be in a mechanism composed of hard rigid tissue."

Duane[6] expresses this action as follows:

"Opposing muscles act not only to check, reverse, or modify the movement initiated by the antagonists, but also by their counteraction make the movement of a moving eye steadier (see pp. 710 and 721), and, furthermore, maintain the *balance* of the eye when at rest. Quite certainly all the muscles take part in this action, so that when the eye is at rest it is maintained in position by the balanced contraction of all six muscles. This is especially the case when no one of the six is predominantly acting, but all are equally relaxed, yet maintain their normal tonus. When this is the case, the eye is said to be in the primary position (or position of equilibrium). In normal subjects each eye is then directed straight ahead in the horizontal plane, the visual axes being horizontal and parallel with the sagittal plane. When otherwise directed, the eye is said to be in a secondary position."

This combined action of the entire muscle force is expressed by Savage[9] as follows:

"In every act of binocular single vision, the triple task imposed on the twelve muscles, by the imperious law of corresponding retinal

points, must be performed. The keeping of the visual axes in the same plane is the chief work of the superior and inferior recti, but they are aided in this work by the obliques, which are also subvertors and supervertors. The intersecting of the visual axes at the point of fixation is the chief, sometimes the only, work of the interni and externi; but the interni are helped by the superior and inferior recti, so that the point of intersection may not be beyond the object; while the obliques assist the externi to prevent the intersection from taking place between the observer and the object. The paralleling of the vertical axes of the eyes with the median plane of the head is the chief work of the obliques, but in doing this work they are hindered rather than helped by the recti. In subversion, the superior obliques aid the inferior recti; but at the same time the former must counteract the mischievous outward torsioning effect of the latter. In superverting the eyes, the inferior obliques help the superior recti, but the former must oppose the inward torsioning effect of the latter.

"Rotation to the right or left in the horizontal plane is effected by all the twelve muscles under the stimulus of seven of the nine conjugate innervations, the subverting stimulus perfectly neutralizing the superverting stimulus, and the *intorting* stimulus just as completely neutralizing the *extorting* stimulus. The axis of rotation is the vertical axis of the eye, and there can be neither elevation, depression, nor torsion while this rotation is being effected. It can hardly be claimed that the conjugate stimulus, which would move the eyes in opposite directions, plays no part; for, if both eyes are to be rotated directly to the right, the internus of the right and the externus of the left must have a control current of nerve impulse sent to them, but a current much less powerful than is sent to the right externus and left internus. This is as necessary as that the conjugate innervation of the subvertors should be neutralized by the conjugate innervation of the supervertors. Thus it would seem clear that in every rotation of the eyes all twelve of the muscles are called into harmonious action under the influence of seven of the nine conjugate innervations, and all in obedience to the law of corresponding retinal points. With mathematical precision, all ocular motions must be accomplished."

The eye moves from one point to another, not alone by the main action of a single muscle and its synergists, but is aided in this motion by the control action of its antagonists, which steadies the eye in its movements and prevents too rapid movements and too sudden a stop with its shock.

Duane[6] gives the action of each muscle in moving the eye about the two primary axes as follows:

"*Abduction.* The eye is carried straight *out* by the external rectus assisted especially toward the end of its course by the two obliques. The latter nullify each other's vertical and torsion action, so that the eye moves straight out and its vertical meridian remains vertical. The external rectus pulls the outer canthus outward.

"*Adduction.* The eye is carried straight *in* by the internal rectus assisted, especially toward the end of its course, by the superior and

inferior recti. The latter nullify each other's vertical and torsion action, so that the eye moves straight inward, and its vertical meridian remains vertical. The inner canthus is pulled back and inward.

"*Elevation.* The eye is carried straight *up* by the superior rectus and the inferior oblique, the external and internal recti probably acting to steady it in its course. The two elevating muscles counteract each other so far as regards lateral action and torsion, so that the eye moves straight up and the vertical meridian remains vertical. The superior rectus helps to lift the upper lid.

"In moving *up* and *out,* the eye is carried up mainly by the superior rectus; out by the external rectus, assisted by the inferior oblique. The vertical meridian is tilted outward.

"In moving *up* and *in,* the eye is carried up mainly by the inferior oblique; in by the internal rectus assisted by the superior rectus. The vertical meridian is tilted inward. (Fig. 30.)

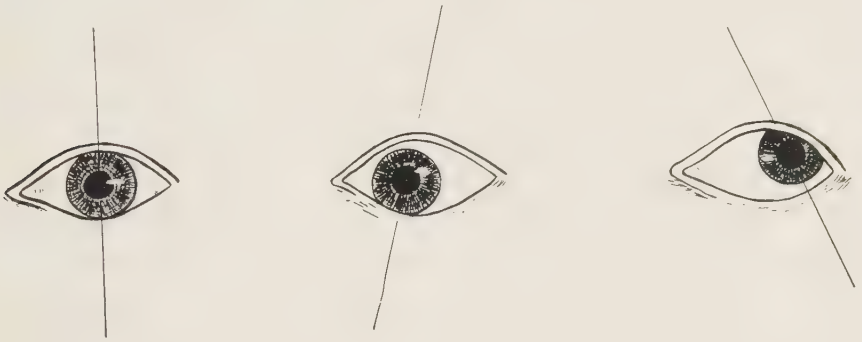

Fig. 30.—Position of vertical meridian of the cornea with different directions of the eye. (Stevens, *Motor Apparatus of the Eyes.*)

"*Depression.* The eye is carried straight *down* by the inferior rectus and the superior oblique, the external and internal recti probably acting to steady it in its descent. The lateral and the torsion actions of the two depressors nullify each other, so that the eye moves straight down and its vertical meridian remains vertical. The inferior rectus assists in depressing the lower lid.

"In moving *down* and *out,* the eye is carried down mainly by the inferior rectus, out by the external rectus assisted by the superior oblique. The vertical meridian is tilted inward.

"In moving *down* and *in,* the eye is carried down mainly by the superior oblique, in by the internal rectus aided by the inferior rectus. The vertical meridian is tilted out."

Binocular Movements

Duane[6] gives the field of action of the various muscles in the cardinal directions, as follows:

"The muscles of the two eyes may be grouped according to their main action as follows:

1. *Lateral Rotators*

Right Rotators	*Left Rotators*
(Carrying both eyes to right)	(Carrying both eyes to left)
R. external rectus	R. internal rectus
L. internal rectus	L. external rectus

II. *Elevators*

Right-hand Elevators	*Left-hand Elevators*
(Acting as elevators mainly when eyes are directed to right.)	(Acting as elevators mainly when eyes are directed to left.)
R. superior rectus	R. inferior oblique
L. inferior oblique	L. superior rectus

III. *Depressors*

Right-hand Depressors	*Left-hand Depressors*
(Acting as depressors mainly when eyes are directed to right.)	(Acting as depressors mainly when eyes are directed to left.)
R. inferior rectus	R. superior oblique
L. superior oblique	L. inferior rectus

"The twelve muscles of the two eyes are thus divided into three groups of four each; and each group comprises two pairs of muscles, one muscle of each pair being in the right eye, the other in the left. The two muscles of each pair are called *associates*.

"It will be seen that each pair of associates has its predominant *field of action* (i.e., direction of the gaze in which it is most active)," viz.:

Eyes directed to—	*Muscle predominantly acting—*	
Right	R. external rectus	L. internal rectus
Left	R. internal rectus	L. external rectus
Up and right	R. superior rectus	L. inferior oblique
Up and left	R. inferior oblique	L. superior rectus
Down and right	R. inferior rectus	L. superior oblique
Down and left	R. superior oblique	L. inferior rectus

The four primary or cardinal directions of gaze are *up, down, right* and *left*. The oblique directions of gaze (i.e., up and right, down and right, down and left, and up and left), are called the *diagonal directions*. The observations of the movements of the eyes in the diagonal directions together with that of the simple right and left movements are of great aid in diagnosing paralysis, and these are termed the *diagnostic directions* (Duane[10]).

There are two distinct binocular movements which deserve attention —*conjugate movements* and *disjunctive*. In directing the eyes in the different fields of regard, the two visual axes are directed toward the same point, the object fixed. These are called parallel movements of the two eyes. In the lateral movement to the right, the eyes are moved by the two right rotators (right external rectus and left in-

ternal rectus) and to the left they are moved by the two left rotators (the right internus and the left externus).

Elevators.—It requires the action of four elevators to move the eyes *straight up,* besides the steadying influence of the external and internal recti, and to keep the vertical meridian straight the two obliques are probably brought into play.

Up and to right requires two right-handed elevators, two left-handed elevators, two right rotators, and both vertical meridians are rotated to the right.

Up and left requires two left-handed elevators, two right-handed elevators, two left rotators, and both vertical meridians are rotated to the left.

Straight down. The two eyes are moved straight down by four depressors (two inferior recti, two superior obliques), steadied in their course by the internal and external recti.

Down and right requires two right-handed depressors, two left-handed depressors, and two right rotators, at the same time the vertical meridians are rotated to the right.

Down and left requires two left-handed depressors, two right-handed depressors, two left rotators, and both vertical meridians are rotated to the left.

Conjugate Innervations.—Maddox,[11] in 1898, stated that the number of known conjugate innervations was five, "of which one elevates both eyes, another depresses them, a third turns both to the right, and a fourth both to the left. The fifth is the converging innervation."

Savage is much more liberal. He claims there are "nine conjugate innervation centers for the control of the twelve extrinsic ocular muscles; that five of these centers are connected with the eight recti muscles, each center with two muscles, one of which belongs to the right eye and the other to the left eye; that four of the nine centers are connected with the four oblique muscles, each center with two muscles, one of which belongs to the right eye and the other to the left eye." It is Savage's opinion that the nine conjugate centers are in the cortex near or in the anterior part of the motor area.

Savage[12] says:

"From each cortical conjugate center go two fibers, or two sets of fibrils; one goes to a single muscle on the corresponding side, the other crossing the median line goes to a single muscle on the opposite side. These muscles, thus united with a common center, constitute a pair. Evidently the centers formed in the cortex of one hemisphere exist in duplicate in the other hemisphere and each duplicate center must have a connection with the same two muscles. To illustrate, the first conjugate center in the left cortex is connected with the two superior recti and supplies them with power. The first conjugate center in the right cortex must have a similar connection with the

superior recti, but this center sends no neuricity to these muscles and therefore does not excite them into contractility."

Savage has given us basal centers for each ocular muscle. Of this he says (l.c., pp. 57-62):

"There is one basal center for each ocular muscle, and each center can act on only one muscle. The basal centers are all under control of the fusion faculty of the mind, and none of them are ever called on to discharge neuricity unless a condition exists that would cause diplopia.

"The basal centers connected with the twelve extrinsic ocular muscles all exist in the interest of binocular single vision; each is connected with only one muscle, and they are all under control of the

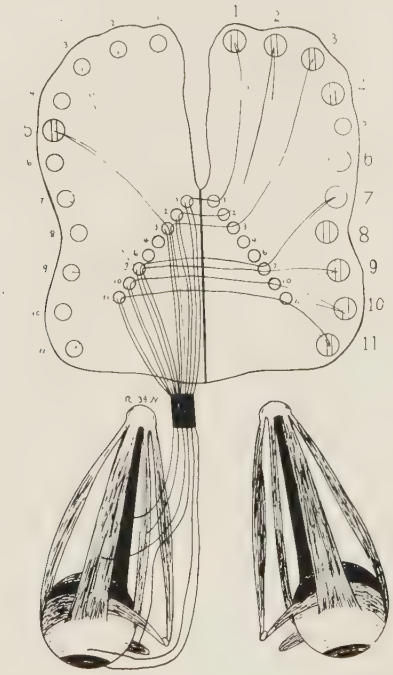

Fig. 31.—The third nerve and its cortical and basal centers. (From Savage, *Ophthalmic Neuromyology.*)

fusion faculty of the mind. Unless some condition exists or arises that would cause diplopia, these centers are ever inactive, their normal state being one of rest. Their location is on either side of the median line beneath the aqueduct of Sylvius and in the anterior part of the floor of the fourth ventricle. In the plates to follow, the basal centers are represented schematically, and they are numbered in harmony with the numbering of the conjugate cortical centers. They exist in pairs, but work independently. They all stand ready always to discharge neuricity that images may be fused but a discharge from a single center can affect only a single muscle.

"A glance at Plate I [Fig. 31] will show that the third nerve cable is composed of insulated fibers from eight of the eleven active conjugate cortical centers; that the fibers from seven of these centers cross the median line to help form the right third nerve, the only non-crossing fibers coming from the fifth conjugate center. The broken lines from the corresponding eight inactive centers are intended to show a connection between these centers and the muscles controlled by the eight active centers.

"Plate I also shows that the right third nerve has in it insulated fibers from six of the eight right basal centers. None of these basal fibers have crossed.

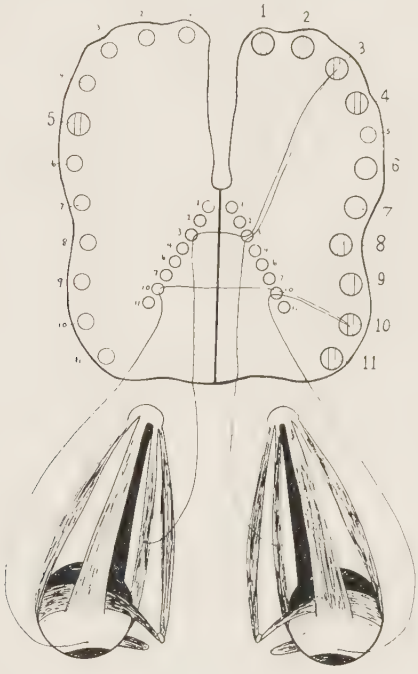

Fig. 32.—Convergence centers. (From Savage, *Ophthalmic Neuromyology.*)

"Plate VIII [Fig. 32] represents the act of convergence of emmetropic-orthoptic eyes * * * the head is still in the primary position, and the near object is in the line of intersection of the extended median and horizontal planes. Volition discharges the third conjugate center and causes a flow of an equal quantity of neuricity to each of the two interni, which, responding with equal power, converge the visual axes to the point of fixation. Simultaneously, volition unlocks the tenth conjugate center, which sends an equal quantity of neuricity to each of the Muller muscles of accommodation, thus causing a perfect focusing, on each retina, of the rays of light coming from the point of fixation." (p. 77.)

Savage has given beautiful schematic representations of the various actions of the different conjugate innervation centers, and the reader is referred to his most interesting books for further details.

Disjunctive Movements of the Two Eyes.—The disjunctive movements of the two eyes are those of *convergence* and those of *divergence.*

Convergence.—When the normal eyes are looking at a distant object, their visual axes are said to be parallel. When these same eyes are required to look at an object nearer, say, than 20 feet, the visual axes are made to converge, and this convergence will depend upon the nearness of the object looked at. This active turning of the eyes inward to focus on a near object is called *dynamic convergence,* and is controlled by the convergence center in the brain. In the normal eye we find no static convergence, as the visual axes are parallel, but in convergent squint there is a static convergence which is equal to the angle of the squint.

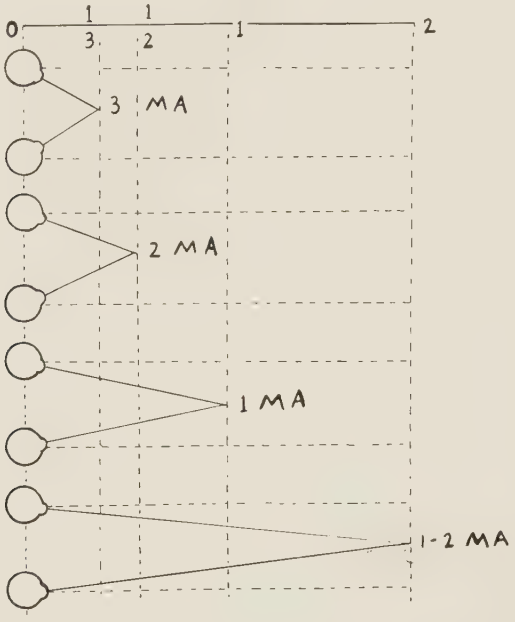

Fig. 33.—Diagram illustrating the unit of convergence, the meter angle. (From May, *Diseases of the Eye.*)

The determination of the convergence near point is very important. (See chapter on Examination of the Patient.) It is the chief means of determining whether or not we have an excess or deficiency of convergence in a given case. In a recent article Duane[13] stresses the importance of accurate measurements and recommends that the measurements should be taken from a point on the nose 9.5 mm. in front of the corneal apex. This same writer insists that in measuring the convergence angle we use the interaxial distance instead of the interpupillary distance.[13] The disturbance of the convergence centers in the brain has much to do with the production of squint.

The nearest point at which the two eyes can fix an object is called the *convergence near point*. The farthest point at which they can be directed by the utmost relaxation of the converging muscles is called the *convergence far point*. This may be beyond infinity and is called negative convergence. Duane claims this condition is normal. If the convergence far point is within finite distance it is spoken of as positive. The *amplitude* of convergence is the distance between the near point and the far point of convergence.

Nagel introduced the meter angle as a measure for the amount of convergence present (Fig. 33). One meter angle is the amount of convergence required when looking at an object one meter in front of the eyes. Thus it will be seen that one meter angle of convergence corresponds to one dioptre of accommodation, as one dioptre of accommodation is required to look at an object one meter distant. This relation holds good at all distances. If an object is looked at when placed 50 cm. away, it requires 2 meter angles of convergence and 2 dioptres of accommodation.

Accommodation and convergence are associated functions but are controlled by separate brain centers, and have certain independence of action, but in daily life, due to heredity and habit, their actions are uniformly associated. The emetropic eye requires as many dioptres of accommodation as it does meter angles of convergence, and vice versa. (See Accommodation.)

Divergence in the Lateral Plane is probably produced by two different sets of innervations acting on two different sets of muscles. First, a relaxation of the two interni, a passive divergence. Secondly, a contraction of the two externi, which is called an active divergence. The normal amount of divergence is about 1.5 to 2 degrees.

Divergence in a Vertical Plane.—This deviation is produced by the simultaneous contraction of the elevators of one eye and the depressors of the other. Duane claims that in the normal eye this is not more than one degree.

Accommodation.—The innate power of the eye to focus an object at different distances is called accommodation. This power resides in the lens and the ciliary body with the muscles and nerves of the latter. Ball[14] describes this function as follows:

"The agents concerned in accommodation are the lens and ciliary muscle. The eye has often been compared to the camera of the photographer: the dioptric media equal the lens, the pigment of the retina and choroid makes the chamber dark, the retina is the sensitive plate, the iris regulates the amount of light, and the focus is obtained by the action of the ciliary muscle on the lens. In the camera the focus is obtained by moving the sensitive plate nearer to or farther from the lens. In the human eye the retina occupies a fixed position, while

the focusing is accomplished by alterations in the curvature of the lens. As regards the exact mechanism of accommodation, authorities differ, there being two principal theories. Helmholtz held that, when the ciliary muscle contracts, the choroid and ciliary is relaxed, and the lens—by virtue of its elasticity—becomes more convex. Tscherning has advanced the view that accommodation does not depend upon slackening of the Zonula of Zinn, but on its tension, which is brought about by the ciliary muscle, whereby the periphery of the lens becomes flattened and the curve of its surface assumes an hyperboloid form. Tscherning states that he has actually produced this change by making traction on the zonula of the eyes of animals from which the cornea has been removed. Schoen has also attacked the Helmholtz theory of accommodation. Which of these hypotheses is correct, must be decided by future investigations."

The nervous center of accommodation is controlled by the short ciliary nerve and the ciliary ganglion, which are derived from the third nerve. The center for accommodation for near objects is located near the front end of the aqueduct of Sylvius. (See Conjugate Centers—Savage.)

Amplitude of Accommodation.—The nearest point at which an object can be distinctly seen or the smallest print can be read is called the *near point* or punctum proximum (abbreviated *p. p.* or *P.*). The most distant point at which an object can be seen distinctly is called the *far point,* or punctum remotum (abbreviated *p. r.* or *R.*). The amount of accommodation which the eye is capable of performing is called the amplitude of accommodation (represented by *A*), and this is usually expressed by the formula, $A = P\text{-}R$, in which *A* represents accommodation, *P* the nearest point, and *R* the most remote point at which an object can be seen. *P* and *R* are usually expressed in dioptres. Where the near point is known in centimeters, the amplitude of accommodation can be reduced to dioptres by dividing 100 by this near point. If the near point is 10 centimeters, we would have the following:

A = 100 ÷ 10 = 10 D. of accommodation, while R equals infinity.

Subnormal Accommodation is divided by Duane as follows:

"(1) Static insufficiency, due to inertia, or lack of response, of the lens to the ciliary muscle.

"(2) Dynamic insufficiency, due to deficient energy of the ciliary muscle, this failure being due to a defective action of the nerves and centers controlling this muscle."

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CHAPTER V

PHYSIOLOGY OF VISION

In order that the student of strabismus may the more fully understand its etiology, pathology, and treatment, we have deemed it advisable to discuss the physiology of vision.

Vision

The sense of sight, the most highly developed of our special senses, is also the most complicated in its action, and possibly the least understood. We shall discuss first some of the elementary optical principles involved in vision; then the retina and its functions, and, lastly, the theories of vision.

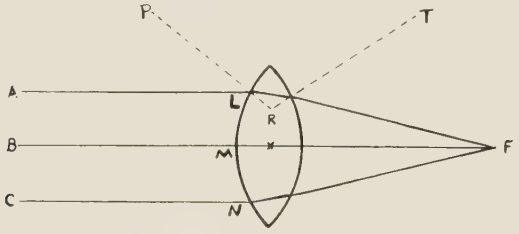

Fig. 34.—The principal focus of a convex lens. (From May, *Diseases of the Eye.*)

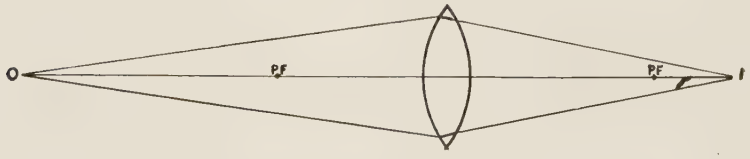

Fig. 35.—Conjugate foci of a convex lens. (From May, *Diseases of the Eye.*)

Refraction of Light.—A light ray passing from one transparent media into another of different density is bent on entering this new media (unless it enters it perpendicular to its surface). This bending of the rays is called refraction.

A Convex Lens.—Parallel rays of light on passing through a convex lens are brought to a point at the focus of the lens. This will depend on the indices of refraction of the glass, and on its two surfaces.

The Foci of Convex Lens.—The point at which rays converge after passing through a convex lens is called its *focus.* The point at which parallel rays come to a point after passing through the lens is called

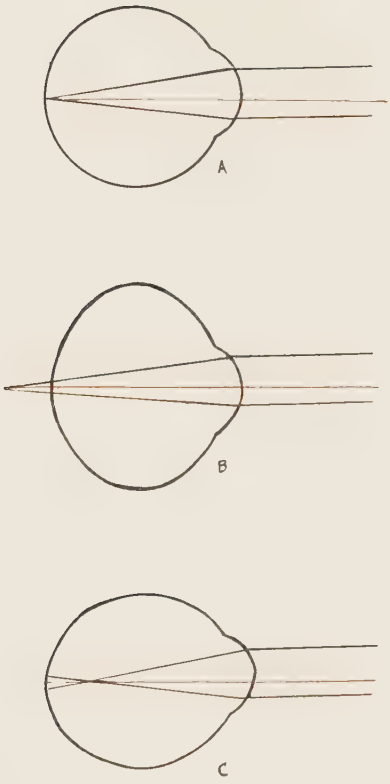

Fig. 36.—*A*, emmetropia. *B*, hypermetropia. *C*, myopia. (*The American Encyclopedia of Ophthalmology.*)

the *principal focus.* The distance of this point from the center of the lens is known as the focal distance. (Fig. 34.)

Conjugate Foci.—These are interchangeable foci in which the image can be replaced by the object and the object by the image. (See Fig. 35.)

Dioptric Apparatus of the Eye.—The eye can be considered as an optical instrument and is at times compared with the camera. By means of its refracting media, small inverted images of objects are formed on the retina, and the impression thus made is carried by the optic nerve and tracts to the cortical center of vision.

The refractive surfaces of the eye are the cornea and the anterior and posterior surfaces of the lens. *The refractive media* are the cornea, the aqueous humor, the lens, and the vitreous; these represent the dioptric apparatus of the eye, and are equal to a convex lens of 23 mm. focus. The refractive indices of each of the above are as follows:

Taking air as a unit 1,000, we have:

Water	1.335
Aqueous humor	1.3365
Cornea	1.337
Lens	1.437
Vitreous humor	1.3365

The drawings in Fig. 36 will indicate the action of the refractive media in different shaped eyes.

Cardinal Points of the Eye.—In order to understand the course of rays of light through the eye, it is necessary to know the cardinal points of the eye. These are as follows, and are graphically shown in Fig. 37 taken from May.

Principal Points are two points so situated that if an incident ray passes through one principal point its emergence ray will pass through the other. These points are situated so close together that for practical purposes they can be considered as one point. They are situated in the aqueous about 2 mm. behind the posterior surface of the cornea.

Nodal Points are two points situated just at the posterior pole of the lens, about 7 mm. behind the anterior surface of the cornea and practically at the optical center of the dioptric system of the eye. They are so close together that they may be considered one point. Rays passing through these points are not refracted.

The First Principal Focus is that point on the optic axis at which parallel rays in the vitreous will meet in front of the eye. This is about 14 mm. in front of the cornea.

Second Principal Focus is a point on the optic axis at which parallel rays of light are brought to a focus by the refractive system of the eye. This is about 23 mm. behind the cornea on the retina, usually between the fovea and the optic nerve.

Visual Acuity is the measurement of the ability to see distinctly. It depends upon the transparency of the refraction media of the eye, the refractive perfection of it, the luminosity under which the objects are seen, the size of the retinal image, the size of the pupil, the nervous functions of the retina and the optic nerve, and last, but not least, the mental ability to make the proper interpretations.

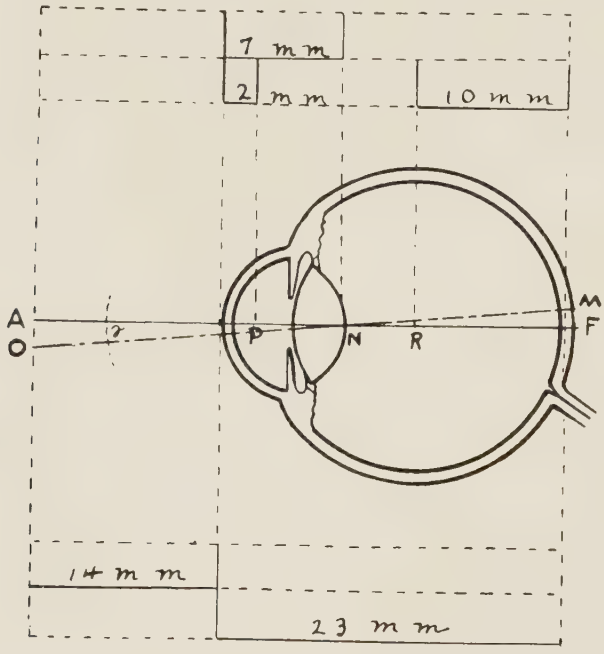

Fig. 37.—Cardinal points of the eye. (From May, *Diseases of the Eye.*)

Visual acuity is measured by the smallest angle under which the eye can distinguish the form of an object. This angle for the normal eye subtends about one minute. (Eldridge-Green gives 50 seconds as normal.) It is also measured by the lowest angle under which two points can be distinguished from each other.[1]

The minimum visual angle varies with the luminosity, the dioptric apparatus of the observing eye, the retina, the optic nerve, and the brain. It is less in youth and more in old age.

An eye which is able to recognize objects subtending only one minute angle is considered to have normal acuity of vision. This varies at dif-

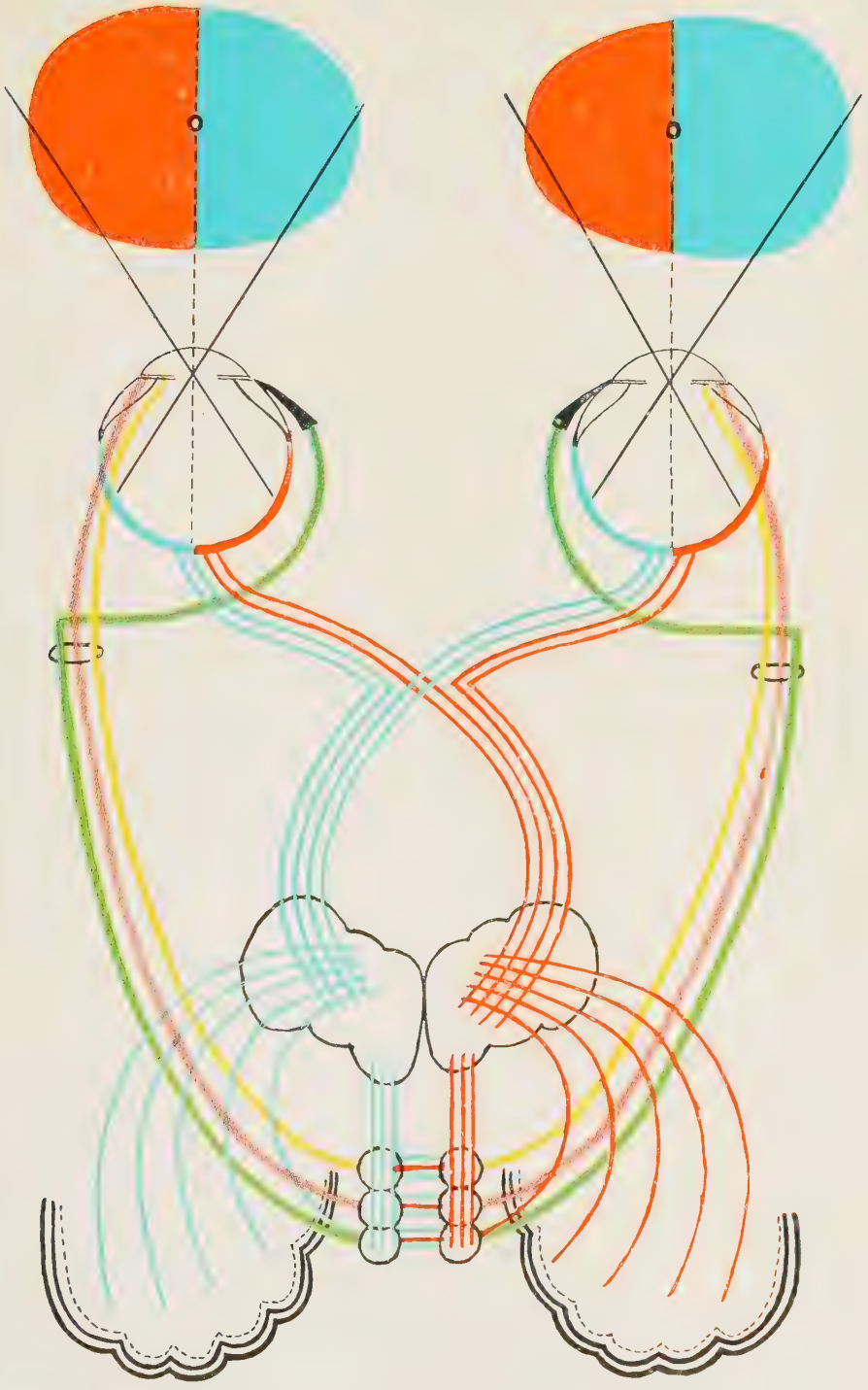

Fig. 38. Schematic representation of the visual and pupillary paths. (From May, *Diseases of the Eyes.*)

ferent parts of the retina. Taking the vision at the fovea as 1, it is only 1/5 at 2° 52′ from the fovea, and decreases gradually. At 10° it is 1/19, and at 30° 20′ it is 1/100.

The Retina

The retina, which is an outgrowth of the brain, consists of a spreading out of the fibers of the optic nerve (Black) over the entire back four-fifths of the eye. This highly specialized nervous structure possesses the power of transforming the inverted image of external objects into a nerve stimulus. This excitation in turn is carried by the fibers of optic nerves and tracts to the visual centers of the brain.

The retina is the receptive layer of the visual apparatus, and this corresponds to the sensitive plate of the camera. The most anterior (front) layer of the retina is the pigment-cell layer. We shall see later its importance in the function of vision. Immediately underneath this layer is the layer of rods and cones. The rods are about 0.06 mm. long and 0.002 mm. in diameter. The cones are about 0.035 mm. long and about 0.006 mm. in diameter.[2]

The Macula Lutea and Fovea is the most sensitive part of the retina. In the macula lutea, or yellow spot, is a small pit which is called the fovea centralis. This point is about 0.5 mm. in diameter. Rods are scarce in the macula and are entirely absent in the fovea. The cones in the fovea are much longer and thinner than found in any other part of the retina.

Functions of the Retina.—The retina has several functions to perform. It receives the stimulation from light and converts it into three senses:

1. Light sense.
2. Color sense.
3. Form sense.

Each of these senses can be quantitatively studied. They vary at different portions of the retina, being more intense at the macula and diminishing toward the periphery. The degree of visual acuity, luminosity, tone, intensity, and saturation also causes marked variations. (Fig. 38.)

These various sensations upon the retina are transmitted by the optic nerve and tracts to the brain, where these sensations are elaborated into perceptions of external objects in some as-yet-unknown manner, and we come to recognize certain objects, with their various colors, dimensions, and relations.

Light Sense.—Physiologists consider that the sensation of light is due to a stimulation of the *rods* and the discrimination between details to the action of light upon the *cones*. The cones predominate in the

macula, whereas, at the periphery of the retina, cones are very scarce; hence, from anatomical deductions, it is inferred that the acute perception of objects is a function of the cones, while light perceptions and observation of moving objects is the function of the rods.

The Field of Vision is the area in which an eye can detect objects while the eye itself is fixed on an object directly in front. This extends upward 60 degrees, outward 80 degrees, downward 70 degrees, and inward 60 degrees. These, of course, vary according to the depth of the eye in the orbit, the overhanging of the upper lid, and the height of the nose.

The Field of Fixation comprises the area in which the eye can fix objects, the head being held stationary. This field is approximately as follows: upward, 35 degrees; outward, 45 degrees; downward, 55 degrees; and inward, 45 degrees. Fig. 39 shows the field of binocular fixation.

Binocular Vision.—When a person with normal eyes looks at an object at a distance, there is a picture of this object formed on each retina at the fovea, and at the same time all near surrounding objects form pictures on each retina at points more or less distant from the fovea. These central foveal pictures are so blended in the brain as to form only one distinct object, and this combining of the two images into one mental picture constitutes binocular vision. This is an innate faculty in man, and is acquired within the first six months. All surrounding objects are seen less distinctly, but not double. This is due to the fact that the surrounding images fall on corresponding retinal points. The right half of one retina corresponds with the left half of the other, and vice versa.

Physiologic Diplopia.—Binocular vision for near objects becomes more difficult than for those at a distance. This is attributed to the fact that objects near the visual axis of each eye do not always fall on corresponding retinal points, and the brain is called upon to eliminate the two images that would otherwise cause diplopia. If a pencil is held in the line of vision when looking at a distant object, two pencils can be observed; but this diplopia, called *physiologic diplopia,* is not ordinarily observed in daily life, due, according to Worth,[3] to the "elasticity of the fusion faculty." Duane[4] is of the opinion that from these physiologic double images we get our "main notion of depth and distance."

Worth[3] gives the following law governing fusion of images:

"When the images formed in the two eyes differ in shape, size, or position, if the disparity be not too great, the oculomotor apparatus first places the eyes in the most favorable relative positions; the fusion sense, by virtue of its elasticity, then fills up any gap which may remain."

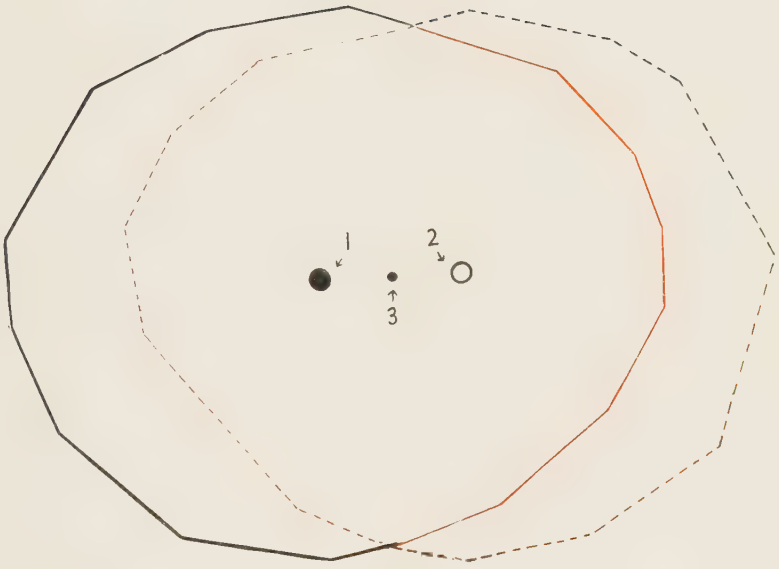

Fig. 39.—Diagram of the field of binocular vision. The field for the left eye is bounded by the solid line; that for the right by the interrupted line. *1, 2,* physiologic scotomata. *3,* fixation point. The part bounded by red lines is the field of binocular vision. (Modified from Ball, *Modern Ophthalmology.*)

Theories of Vision.—Vision is a sensory stimulation, a very intricate process, of which there are many theories, all of which fail in some way to definitely and satisfactorily explain all the phenomena that take place from the emission or reflection of light by an object to its perception on the retina, and its proper interpretation by the brain. These various steps can best be divided into:

(a) *Problems in Optics.* The formation of the image of the object on the eye.

(b) *Nerve Condition.* The passage of this impulse created by this image on the retina to the brain.

(c) *Brain Physiology.* The reception of this impulse and interpreting correctly the sensation.

The mechanism of vision is well stated by Bayliss[5] as follows:

"There is every reason to believe that the means by which light stimulates nerve endings is through a photochemical reaction. There are an enormous number of chemical reactions which are affected by light, and, of these, one is known in connection with the retina, namely, the changes in the visual purple. Whether this is the only one, we cannot say with certainty, but we shall see presently that its properties are in extraordinary coincidence with certain aspects of vision. If a substance is sensitive to rays of a particular wave length, such as would be necessary to account for color vision, it must absorb these rays. Since they must be in the region of the visible spectrum, the substance must have an absorption band in the region referred to, and, therefore, be possessed of color itself. Although visual purple is the only such substance detected as yet in the retina, with the exception of certain colored globules, which are not sensitive to light, described by Kuhne (1878), in the cones of birds, it is conceivable that others may be present in the small amounts required, and even in the requisite number to account for the number of colors to which the eye is sensitive.
* * *

"Visual purple is found in the rods only of the mammalian eye, in the so-called cones of birds, and in the corresponding structures of the retina of the frog, fish, and cephalopod. Since it is not present in the cones of the human eye, it is absent from the region of sharpest vision, the fovea centralis; a fact which has led some observers to doubt whether it has any function in day vision. Eldridge-Green supposes that it diffuses into the fovea from the surrounding rods. The rods themselves, he regards as being concerned only with the formation of the pigment and not receptor organs for light. As regards this last point, it appears that the sensibility of the various zones of the retina in the recognition of form during day vision is directly proportional to the number of cones per unit area which they contain. Put in another way, the images of two points are recognized as distinct according to whether or not they fall on two cones which have between them an unstimulated cone. So that they must be further apart to be recognized as two in the peripheral parts of the retina, where the cones are further apart.

"When light falls on the peripheral parts of the retina in man, it

is found that, when diminished so as to be just visible, it is only that part of the spectrum between wave lengths 600 and 440 (orange and blue) that is visible at all, and the sensation is one of light only, without color, whatever the wave length used. * * *''

Color Sense.—Color vision is both physiologic and psychologic. Various hypotheses have been present to explain the mechanism of color vision. The Young-Helmholtz theory[6] is as follows:

''In this theory it is postulated that each nerve fiber of the retina is composed of three submembers or fibers, each of which is provided with a special terminal organ (a photochemical substance). An irritation of the first fiber supposedly produces a violet sensation, an irritation of the second fiber a green sensation, and of the third, a red sensation. These three are the primary or principal sensations giving rise to the principal colors. An irritation, then, of the red and green sensation fibers would produce yellow, and so on through the color scale. White is produced by the simultaneous irritation of all three fibers; no irritation of any of the fibers gives the sensation of black.''

One of the most interesting and recent theories is the Ladd-Franklin theory,[7] in which ''A primitive photochemical substance, which is composed of numerous gray molecules, is assumed as being responsible for the colorless sensations of white, gray, and black.''

These molecules exist in the primitive state only. The breaking up or dissociation of the molecules causes the sensations of the various colors.

Light stimulus on the retina is recognized by the brain as visual sensation. The processes by which ether-wave impulses cause visual impulses is still obscure. They are usually ascribed to (1) chemical action, (2) molecular strain, (3) electrical action.

The Chemical Theory[8] maintains ''that certain visual substances in the retina are affected by light and that vision originates from the metabolic changes produced in these visual substances.'' The above theory is complicated and not altogether satisfactory.

Eldridge-Green[9] says:

''A ray of light impinging upon the retina liberates the visual purple from the rods and a photograph is formed. The rods are concerned only with the conveyance of the light impulses to the brain. The ends of the cones are stimulated through the photochemical decomposition of the visual purple by light, and a visual impulse is set up which is conveyed through the optic nerve fibers to the brain. The character of the stimulus and impulse differs according to the wave-length of the light causing it. In the impulse itself we have the physiological basis of the sensation of color.''

The Molecular or Mechanical Theory[10] is explained as follows:

''It is readily conceivable that a ray of light can cause a chemical decomposition of a substance in which the rhythmic excursions of an atom or atoms from the center of attraction in a molecule are in

exact tune with the waves of light falling on such atoms. The excursions may be so increased in extent by the rhythmic energy supplied by the light waves that the atoms will leave the parent molecules and produce new molecules."

The Electrical Theory claims that the visual impulse is the concomitant of an electrical impulse.[11]

"* * * that an electrical current is generated in the retina under the influence of light and that this is transmitted to the brain through the optic nerve. It is an undoubted fact that light gives rise to retinal currents and that, on the other hand, an electrical current, suitably applied, causes the sensation of light. Holmgren, Dewar, McKendrick, Kuhne, Steiner, Waller, and others have shown that illumination produces electric variation in a freshly excised eye."

Fritz Schanz's Theory. A very interesting theory of vision which might be called the electron theory is given as follows by Fritz Schanz:[12]

"If two identically equal metallic plates are put in an electrolytic solution and the two plates are connected, an electric current is produced when the one plate is illuminated and the other remains in the dark. The electric current is increased when a dye, known to be a sensitizer, is added to the electrolyte. The same process, I am convinced, is the basis of all sensitization processes in living matter. These investigations give us insight, therefore, as to why the sun acts as motor in the machinery of all earthly life.

"If these observations relative to the elementary actions of light in Nature are correct, then the same process must be the basis of the visual act. Light can act only when it is absorbed. We have considered the rods and cones to be the elements sensitive to light. We are to conclude, therefore, that they are unable to equally absorb the visible light rays. These light rays are, however, absorbed by the pigment-epithelium of the retina. We have, then, the right to surmise that electrons are emitted because of the light absorbed in the pigment-epithelium in the same way as from the numerous pigments examined. The rods and cones of the retina act then as the receiving electrodes which capture the electrons in a manner analogous to the investigations on photoelectric effects. Electrons of different velocity are emitted by light of different wave lengths. The stimulation set up in the rods and cones is conducted through the retina and optic nerve towards the central receiving organism. We can measure these effects in the optic nerve as the currents of response."

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[4]Duane, A.: Fuchs' Textbook of Ophth., ed. 6, p. 732.
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[7]Amer. Ency. of Ophth., xiii, 9997.
[8]Amer. Ency. of Ophth., xiii, 10009.
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[10]Amer. Ency. of Ophth., xiii, 10009.
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CHAPTER VI

TYPES OF STRABISMUS

Strabismus is usually divided into paralytic and comitant. Paralytic squint is a pathologic condition, and is due to a lesion of the nerve supplying the muscle affected. Paralytic squint will be discussed in a separate chapter.

Comitant squint is a functional disturbance, and is usually divided into periodic, constant, unilateral (or monolateral), and alternating. Periodic squint is divided by Worth into premonitory occasional and true occasional, which he places under the main heading of occasional squint. Alternating squint he divided into accidental alternating and essential alternating. The well-known division of squint by Stevens described in Norris and Oliver[1] under heterotropia, which is a general term, includes all forms of deviation of the visual axis from the object fixed by the master eye. He divides these as follows:

Esotropia is a deviation of the visual axis inward.

Exotropia is a deviation of the visual axis outward.

Hypertropia is a deviation of one visual axis above that of the other.

Anotropia is a deviation of the visual axes of both eyes above the normal plane of adjustment.

Katotropia is a deviation of the visual axes of both eyes below the normal plane of adjustment.

Cyclotropia, introduced by Savage, is a deviation of the vertical axis of the eyes, either diverging from above, which is plus-cyclotropia, or converging from above, which is minus-cyclotropia. It can be readily seen that more than one form of these tropiae may be observed in the same person.

The following classification of squint, alphabetically arranged, is taken largely from the American Encyclopedia of Ophthalmology:[2]

Strabismus Absolute.—A deviation occurring at all distances.

Strabismus Accommodative.—This is a name given to squint which is markedly increased when the object is made to approach the eye. Practically all accommodative squints are at first periodic, and usually intermittent. Accommodative squint is produced by excessive effort at accommodation and usually is associated with hypermetropia; however, it is found in deficient efforts made in myopia. This form of squint is usually relieved by the correction of the hypermetropia. Donders considered that practically all cases of comitant squint were due to accommodation associated with hypermetropia. This is the dominating cause in about 30 per cent of cases.

Strabismus Alternating.—In this condition first one eye fixes, and then the other. These cases usually are periodic at first, and finally become permanent. The vision in each eye is usually normal. This type of squint is of relative frequency, and is often associated with accommodation excess. It is in this type of squint that Worth finds his essential squints.

Strabismus Apparent.—Owing to the size of the angle gamma (sometimes called alpha), there is frequently an apparent excess of convergence in myopic eyes and divergence in hypermetropics. Apparent squint may also be caused by the shape of the nose, or rather the overlapping of the skin and the inner angles of each eye. This is often found in the Mongolian race.[3] This condition was first described by Johannes Muller[4] under the name of **Incongruous Strabismus.**

Strabismus Bilateral.—The name sometimes given for strabismus affecting both eyes alternately.

Strabismus, Braid's.—This is a turning of the eye simultaneously upward and inward. This method is sometimes used by hypnotists in order to more readily hypnotize their subjects.

Strabismus Comitant.—This is a general division of squint as opposed to paralytic squint. The nonfixing eye follows or accompanies all the movements or excursions of the fellow organ. In paralytic squint, there is a limitation of excursion and the deflecting eye does not accompany the sound eye in each field of regard.

Strabismus Concomitant.—An older term for comitant squint.

Strabismus Convergent.—In this condition the deviating eye turns in, causing convergence of the visual axis. This is the type most frequently seen, and particularly so in the comitant variety, in young subjects.

Strabismus Deorsumvergens.—This condition is known also as descendant squint[5] and hypertropia[1] downward squint. It is a condition in which the eyeball is turned below the visual axis, and is usually associated with internal or external squint.

Strabismus Divergent.—This is a condition in which the eyeball turns outward, causing a divergence of the visual axes. It is usually associated with myopia, and most frequently occurs during early adult life in contradistinction to convergent comitant squint, which occurs usually in children about three years of age.

Strabismus Dynamic.—A tendency to squint which is overcome by an effort (esophoria).

Strabismus Essential.—This is a type of squint which is found in alternating cases and which is defined by Von Graefe as "antipathy

to binocular vision.'' Savage claims that no amount of effort can ever make the two images fuse in a case of essential squint, as, in his opinion, the two centers for fusion are not neurologically connected. Stevens[6] considers this condition due to declination, and that it usually is associated with a declination produced by faulty operations.

Strabismus External.—(See Strabismus Divergent.)

Strabismus Fixus.—A fixed strabismus.

Strabismus Functional.—Comitant strabismus (Landolt).

Strabismus Incongruous.—(See Strabismus Apparent.)

Strabismus Insuperable.—A condition in which the patient is not able to prevent squinting for near objects, but is able to maintain binocular vision for distance (Maddox[5]).

Strabismus Intermittent.—A squint which under the same condition of vision is sometimes present, and sometimes not.

Strabismus Internal.—(See Convergent Strabismus.)

Strabismus Latent.—That which occurs under special conditions, is usually associated with emotion, and is related to heterophoria.

Strabismus Manifest.—Strabismus occurring when both eyes are open.

Strabismus Mechanical.—This form of squint is due to pressure or traction upon the eyeball, and is usually associated with a tumor or some other pathologic conditions in the orbit.

Strabismus Monocular.—This is a type of squint in which deviation is definitely located in one eye. (See Unilateral Strabismus.)

Strabismus Noncomitant.—(See Paralytic Strabismus.)

Strabismus Occasional.—Worth[7] has divided occasional squint into two varieties—premonitory occasional and true occasional. Occasional squint he defines as an eye which occasionally deviates from the normal relative direction. In this case the squint is seen for only a few seconds at a time, and finally the eyes become more and more frequently crossed. He uses the word ''occasional'' in preference to the word more frequently used in this country, ''periodic,'' which he considers should be used for squint appearing on alternating days, as a tertian ague. Premonitory occasional squint is a type of squint which may occur at very infrequent intervals, and finally disappear entirely, or may maintain this characteristic throughout life.

Strabismus Paralytic is due to a paralysis of one or more of the ocular muscles, and is a general term opposed to comitant squint.

Strabismus Periodic.—This is an occasional squint, and is generally a forerunner of permanent squint, and is usually associated with excessive accommodation.

Strabismus Permanent.—This is a form of deviation which is constant in one or the other eye. This type of squint may be either permanent alternating or permanent monocular. The latter is the ordinary form of comitant squint seen in children after the defect has been present for some time.

Strabismus Relative occurs only when the object looked at is at a certain distance.

Strabismus Spasmodic.—(See Strabismus Spastic.)

Strabismus Spastic is due to spasms of one of the ocular muscles. This form of squint is very rare, and includes some cases of intermittent comitant squint, choeric squint, or irritating cerebral lesions and hysteria and epilepsy.

Strabismus Spurious is the squint seen in young children before the fusion faculty has made much progress in development (Worth[7]). In this squint the eyes converge for a few seconds at a time.

Strabismus Superable is the condition in which the patient is able to maintain binocular vision for distance but squints for near. (See Strabismus Insuperable.)

Strabismus Suppressed.—Akin to latent squint, and expressed by the word "heterophoria."

Strabismus Sursumvergens.—The deviation of the eyes upward, also known as vertical strabismus, or strabismus ascendens (Maddox).

Strabismus Unilateral.—Strabismus affecting one eye. (See Monocular Strabismus.)

Strabismus Vertical.—(See Strabismus Sursumvergens.)

Duane,[8] in Fuchs, has classified squint from an etiologic standpoint as follows:

"A. An anomaly of structure, insertion, or innervation of one or more of the *ocular muscles* (muscular paralysis and spasm, using these terms in their widest sense).

"B. Anomalies of the *coordinate movements of the eyes* (the muscles per se being normal). These include:

"1. Anomalies of *convergence* and *divergence,* either in the sense of excess or insufficiency.

"2. Anomalies of *conjugate movements* comprising conjugate paralysis and spasm, nystagmus, and dissociated movements."

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CHAPTER VII

MEASUREMENT OF STRABISMUS—STRABISMOMETRY

The importance of the accurate determination of the degree of squint cannot be too strongly impressed upon the reader It aids in determining the type of squint and its etiology, and it guides our future treatment of the case; but its chief importance lies in the fact that it is the *one* and *only* guide as to when and upon whom to operate.

Definite repeated measurements of the degree of squint for 6 meters and 60 centimeters should be taken, both under atropin and without it, repeated (without atropin) at weekly or biweekly intervals, and careful records kept of the measurements. This will give us information which can be obtained by no other means. A squint which does not decrease under the use of atropin for a few weeks is not apt to be relieved by the use of glasses, and the converse is true. A case of recent periodic squint, or any case of recent squint in which the squint disappears under atropin, will very probably be relieved by the full correction of the refractive defect. This is usually what occurs when we can see these cases early.

Constant squint, where the vision of the deviated eye is good, and which is lessened by the use of glasses, pads, and atropin, after a few months' treatment will, in all probability, be cured without an operation. Any decrease in the degree of squint is always a hopeful sign, and a continuation of the nonoperative treatment is indicated.

The great importance of accurate measurements must be evident, notwithstanding the fact that Javal[1] says: "It is rarely useful to measure strabismus exactly." This very remark itself is proof of the importance of repeated accurate measurements, as it becomes diagnostic. In a case in which there is a marked difference in the degree of squint, as is indicated by Javal's remark, we would expect to find a recent squint in an individual of an emotional, nervous temperament.

If at any age the degree of squint is not decreased after four to six months of careful attention, it becomes an operative case. If we have been careless in our measurements and still less careful with our records, we cannot intelligently advise as to the future treatment. Accurate measurements for both far and near are diagnostic as to cause, and in operative cases determine largely the operative procedure to follow. Dunnington,[2] discussing operative treatment of squint, says:

"Estimation of the amount of deviation present at both twenty feet and thirteen inches tells us whether the squint has been caused

by an anomaly of divergence or convergence. A deviation most pronounced at twenty feet means primarily an anomaly of the diverging power, whereas, one that is greatest at thirteen inches denotes an error of convergence.''

That the method of measuring squint has not been entirely satisfactory is evident from the many methods to be found in literature. We shall present several of the best known methods, stressing their advantages and disadvantages.

Linear Strabismometry.—The linear measurement of squint was at one time very popular, and was the chief method in use until the angular measurement was introduced by Landolt. Lawrence devised a strabismometer, which is still in use.

Those seen at the present time consist of a flat piece of ivory, concave on one side, to fit the lower lid, with scales in millimeters read-

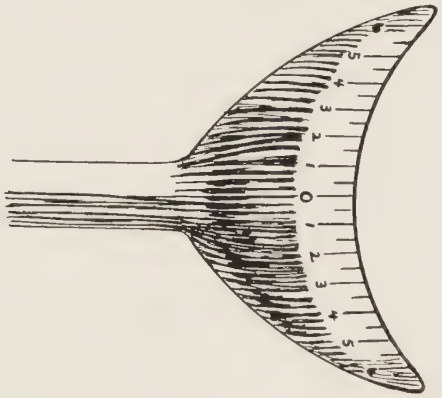

Fig. 40.—Strabismometer. (From Ball, *Modern Ophthalmology.*)

ing from a central zero, both to the right and to the left. It is to be placed upon the lower lid of the patient, sitting in front of a window, while he is looking at some distant object with the deviating eye; the zero on the scales is placed on the lower lid directly underneath the center of the pupil; the good eye is now uncovered and the squinting eye will turn in or out, and the figure which now lies directly underneath the pupil of the squinting eye will measure the amount of deviation. The evident inaccuracies of this measurement render it practically useless.

Galezowski's strabismometer consists of a frame on the order of a trial frame, with adjustable needles or pointers placed on a bar, the latter having scales.

Hirschberg's Method.—Since the breadth of the cornea is about 12 millimeters, and one-half of that length being 6 millimeters, Hirschberg calculated that if a light were reflected into the eye, the image

falling on the edge of the cornea would represent an angle of 45 degrees, and an image falling one-half of the distance between the center of the cornea and its image would represent a deviation of 22½ degrees. The two deviations on which he laid particular stress were the corneal edge, which represented 45 degrees, and the pupilar edge, which represented 15 degrees. This method is but little more accurate than the strabismometer of Lawrence.

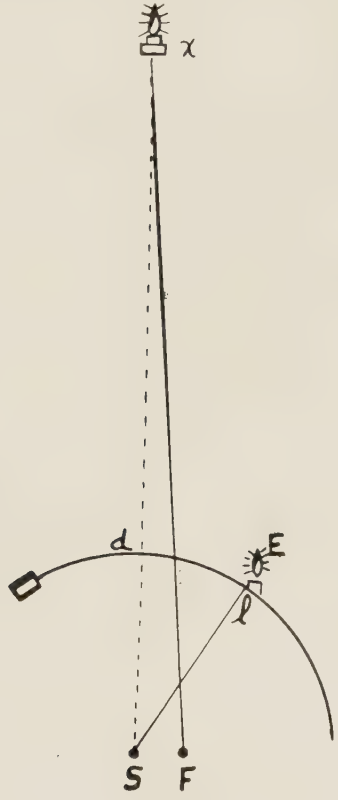

Fig. 41.

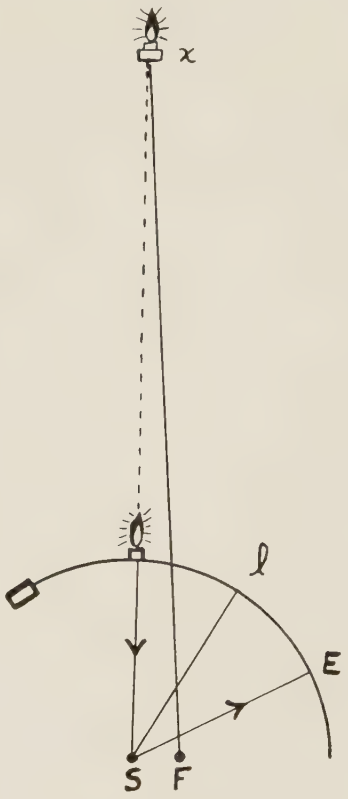

Fig. 42.

Fig. 41.—Javal's method of measuring strabismus.
Fig. 42.—Charpentier's method of measuring strabismus.

Angular Measurement.—This measurement of squint was first suggested by Landolt and the use of the perimeter for determining the amount of deviation of squint was recommended by Javal. Figs. 41 and 42 and description, taken from Maddox, explain the method of Javal and that of Charpentier:[3]

Javal's Method.—"The patient should be seated so as to bring the squinting eye (see Fig. 41) into the center of the perimeter, while straight in front, at a distance of five meters, is placed a candle for the fixing eye (*F*) to look at. It is only some perimeters which permit this.

"Another flame is then moved along the arc of the perimeter, with the surgeon's eye ever behind it, till its reflection appears to occupy the center of the cornea, or rather that part of the cornea which our knowledge of the angle gamma leads us to select, and which I have called elsewhere the 'fixation position.' The squint is now measured by that figure on the perimetric arc which lies nearest the flame.

"For greater accuracy the angle gamma can be measured separately by screening the good eye and making the squinting one fix the ivory disc of the perimeter, while a flame (with surgeon's eye kept strictly in line behind it) is moved along the arc till its reflection appears to occupy the exact center of the corneal circumference. The figure reached by the candle enables the angle gamma to be at once read off from the perimeter. This angle should be subtracted from the record of a convergent squint and added to that of a divergent, premising, of course, that they have been measured by the center of the cornea.

"In figure '*d*' is the ivory disc of the perimeter, and, were there no squint, the visual axis of the left eye would pass through this, as shown by the dotted line. The angle *d S l*, therefore, is the angle of the squint, and is measured by the arc *d l*.

"By bringing the distant flame nearer to the perimeter, the squint can be measured under different accommodative conditions; or, finally, the ivory disc may itself be made the object of fixation.

"The figure makes evident, also, how in a squint of low degree the method is rendered impracticable by both flame (*E*) and the surgeon's head behind it, interfering with the vision of the distant flame by the fixing eye (*F*)."

Charpentier's Method.[3]—"The difficulty just spoken of is evaded in the plan illustrated in Fig. 42 where advantage is taken of the law that angles of incidence and reflection are equal. The flame is placed over the fixation spot of the perimeter, and the surgeon's eye is made to travel along the arc till its reflection appears to lie in the center of the cornea of the squinting eye. The squint is then measured, its angle being *half* the angle of the arc."

The Schweigger hand perimeter is one of the best perimeters for measuring squint (Fig. 43). It is adapted for measurement at 6 meters and 60 centimeters, and can be used with advantage with children, as it can be held by an assistant or nurse.

Angle Gamma.—All perimetric measurements of squint must take into account the angle gamma.

The angle gamma is the angle formed by the intersection of the visual axis with the optic axis. The visual axis is a line beginning at the *fovea centralis retinae* and passing through the center of motion and thence through the cornea (at no definite point) and thence to the object of attention. Visual axis is the anteroposterior axis of rotation. The optic axis is a line beginning at the center of the cornea and passing through the center of rotation and extending to the retina, near or at the macula. This line usually does not fall upon the macula, but falls to one or the other side of it. The angle between

these two lines is known as the angle gamma. (The angle gamma is often confused with the angle kappa.)

The only point necessarily common to these two lines is the center of rotation of the eye. When this angle is to the nasal side of the visual axis, the eye would appear to deviate inward; if to the temporal side, it would appear to deviate outward. It is more often temporal than nasal. The manner of measuring this angle is given by Duane[4] as follows:

"The angle gamma is determined sufficiently well by placing the eye at the center of a perimetric arc and making it fix the zero point

Fig. 43.—Schweigger's hand perimeter. (From Maddox, *The Ocular Muscles.*)

of the perimetric scale. A light is then carried along the arc until its reflection occupies the center of the cornea. The position of the light on the perimetric arc shows the amount of the angle gamma."

A better perimetric measurement is to use the Charpentier method, as follows:

The eye being placed in the center of the perimeter, it is directed at a distant object over the fixation point on the perimeter, while a light is held directly underneath the fixation point, and the surgeon's eye is made to travel on the arc until the reflection of the light lies in the center of the cornea. The number on the perimeter at this

point represents double the amount of the angle gamma. The advantage of this method of measurement is due to the doubling of a small angle, thus lessening any error in its measurement.

Fig. 44-*A*.—First step in tangent strabismometry; adjusting distance of patient by metro-string. (From Maddox, The Ocular Muscles.)

Fig. 44-*B*.—Second step; examining the reflections. Also estimating the vertical element. (From Maddox, *The Ocular Muscles.*)

Maddox's Tangent Strabismometer.—This is a very popular method with many of the English ophthalmologists. It consists of numbers so placed on a wall at one meter's distance in front of the patient

that they represent, respectively, a degree of deviation.[5] Fig. 44 *A* and *B* and description from Maddox[3] are sufficiently explanatory:

"Placing the patient facing the candle at the meter distance, the surgeon introduces his own head between the two, but a little lower down, about a foot away from the patient and so that the root of his own nose is vertically under the rays of light which proceed to the patient's eyes. At once the tell-tale corneal reflections reveal which eye is the squinting one, and the degree of squint being guessed at by the degree of eccentricity of the reflection, the patient is told to look at the figure which numerates the guess.

"If the guess be true, the squinting eye has been brought straight for the candle, and the reflection upon it occupies its proper position.

"If the guess be only partially correct, successive figures are mentioned, one by one, for the patient to look at, till the surgeon is satisfied as to the right one. For rapid work, this suffices, and takes scarcely more than half a minute. Since the height of the patient is immaterial, no time is lost in adjusting it, as in the use of the perimeter."

This method is relatively accurate and has two great advantages: first, it is always ready, and, second, it is a great time saver.

The Priestly Smith Tape Measure.—This method was published in 1888 and is, with some modifications, one of the most accurate and most popular methods at the present time. It is particularly serviceable in the measurement of squint in young children and patients with amblyopic eyes. It is described by Maddox[3] as follows:

"A piece of tape one meter (or 60 cm.) long, of which one end is held by the patient against his temple while the other end is attached to a ring on the surgeon's finger, maintains the requisite distance between the surgeon and the patient. A second piece of tape, graduated and figured, is attached by one end to the same ring, and then passes between the fingers of the surgeon's free hand, at which the patient is directed to look. When the separation of the surgeon's hand reaches the measure of the squint, the corneal reflection occupies the normal position of the cornea of the squinting eye."

Holzer[6] has made a slight, though a very important, modification of Priestly Smith's tape measure which I have used with increasing confidence during the past five years. Holzer describes his method of using the tape as follows:

" * * * Let us take, for example, a convergent squint of the left eye. Measurements should be made in a dark-room with a lamp situated behind the patient. (Fig. 45.) The ring *A* is held against the right cheek below the fixing eye by the patient. An ophthalmoscope or retinoscope handle is passed through ring *B* and held in the observer's left hand. The image of the light behind the patient is now reflected from the mirror of the ophthalmoscope or retinoscope into the fixing eye, and the patient is directed to fix the mirror. Through the sight-hole in the mirror the observer notes the exact position of

the corneal image. He now turns the mirror to the left eye, the patient still fixing the mirror with the right eye, and notes that the image on the left cornea is situated eccentrically outward, showing that this eye deviates inward. He now directs the patient to follow with his fixing eye a finger of the observer's right hand over which ring *C* has been placed. The right hand is gradually moved horizontally outward along the tape *B C* while the observer notes the changing position of the corneal image through the sight-hole of the mirror until the left eye has rotated outward enough to bring the image to the *corresponding situation* occupied in the fellow eye (right) when the eye was

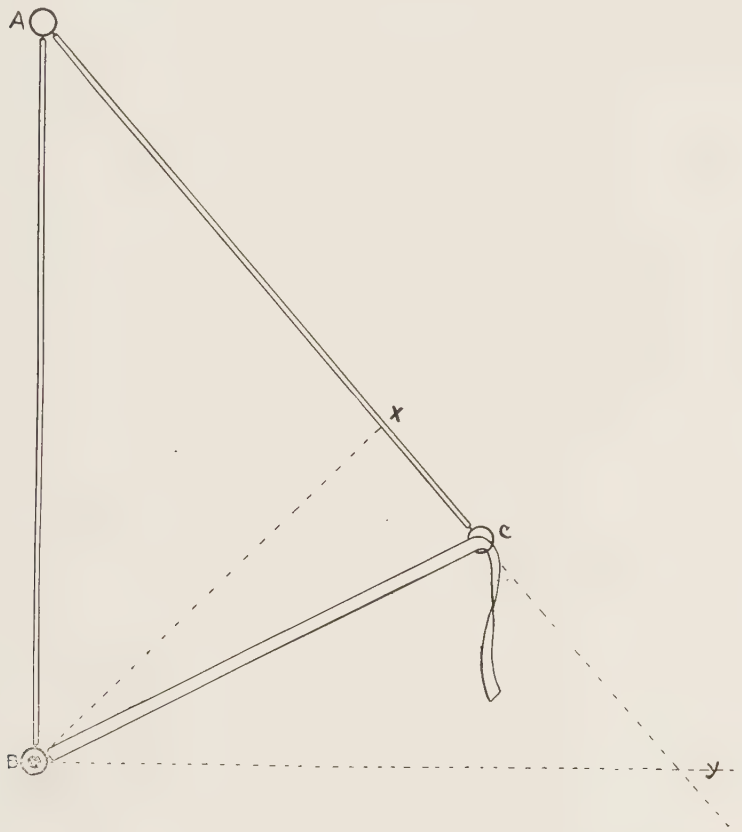

Fig. 45.—Holzer-Priestly Smith tape. Position of tapes in determining squint of 45 degrees, showing advantage of additional tape *AC*. (Holzer) This prevents errors from holding tape *BC* too near patient (*X*) or too far away (*Y*). (*The American Encyclopedia of Ophthalmology.*)

fixing the mirror. When this point is reached he pinches the tape and reads off the degree of deviation; one centimeter on the tape equalling one degree of deviation.''

Some time ago the writer devised the following modification of the Holzer-Priestly Smith tape for measuring the degree of deviation for distance. This is particularly useful in measuring the squint in very

young children for both near and distance. It consists of a very light piece of wood, 60 cm. long with one end so shaped as to fit the cheek under the eye, and a ring one inch in diameter is placed at the other end. Just in front of this ring is a clip to hold pictures when measuring the deviation for near. In the center of the end bearing the ring is fastened a tape graded in centimeters. Twenty centimeters from the cheek end of the piece of wood is attached a handle (see Fig. 46). At the center of this same end and underneath is fastened a piece of tape 60 cm. long with a small ring at the other end. A candle or light is placed at 6 meters in front of the child. The instrument is handed to him and he is directed to place the cheek end of it under his straight eye while he looks at the light through the ring at the other

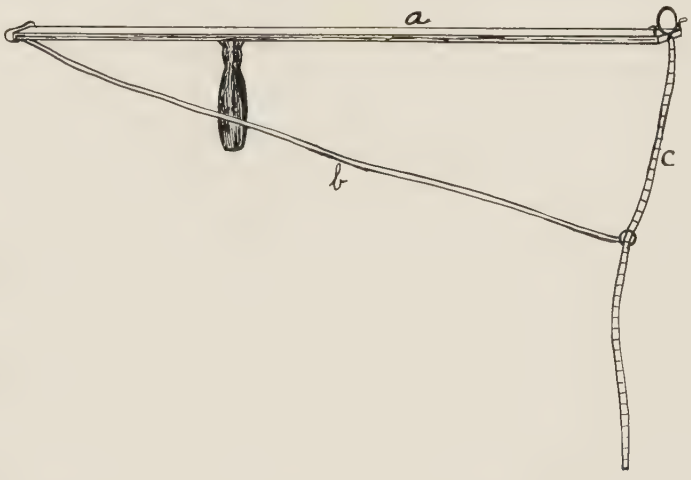

Fig. 46.—Wilkinson's modification of the Holzer-Priestly Smith tape. *A*, piece of wood 60 cm. long with cheek piece at one end and ring at other to sight through. In front of the ring is a clip to hold pictures when using for near. *B*, tape 60 cm. long, one end of which is attached at the cheek piece and to the other end is attached a small ring. *C*, tape graded in centimeters attached to the piece of wood at the sighting ring which passes through the small ring on the tape, *B*.

end. (The instrument can be supported by an assistant when necessary.) The surgeon now takes the ring on the end of the cheek tape, places the graded tape through it, and holds it in one hand. Holding his electric retinoscope in the other hand, he so places his eye and the retinoscope as to cause the corneal reflex to fall in the center of the pupil of the deviating eye. The tape is drawn out taut and the number of centimeters on the tape over which the eye is sighted will indicate the degree of deviation. In very young children the mother can be used as the object looked at and placed at a greater distance, and fairly accurate results will be obtained. Where the deviation is very marked or the nose high, it is advisable to turn the patient's

head so as to partially straighten the deviated eye in order to turn it out of the nasal angle.

Worth, of London, has combined Maddox's tangent scales and Priestly Smith's tape into a strabismometer, which he has called a deviometer (Fig. 47). This is a most excellent and accurate instrument, but not as convenient as the Smith-Holzer tape. One advantage of the last two methods is that they take care of the angle gamma more accurately and with less trouble than can be done with the perimeter. These methods are more convenient in the measurement of squint in children. This is done by having the patient fix the light first with the straight eye, noting accurately the relation of the

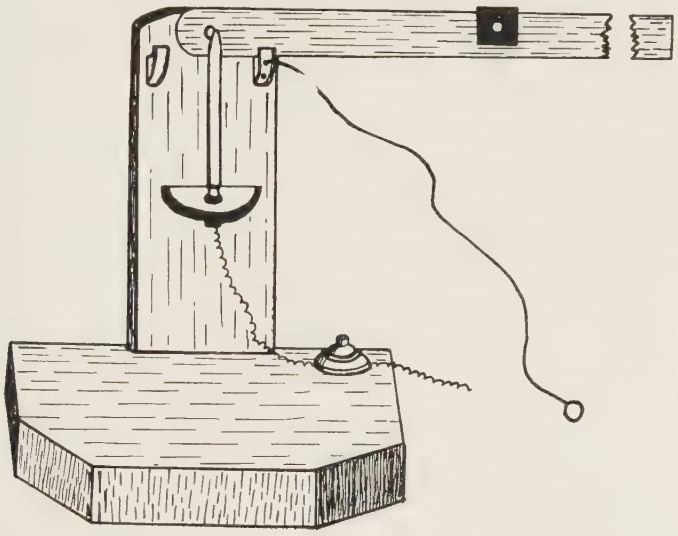

Fig. 47.—Deviometer. (From Worth, *Squint.*)

corneal reflex with reference to the pupil. The tape is moved until the deviated eye assumes the position of fixation, and the image on this deviated eye is caused to fall on the same relative position of the deviated eye as was noted upon the straight one.

Subjective Measurements.—The subjective measurements of squint are without doubt the most accurate methods. Their faults lie in the inability to use these methods in those cases in which there is a want of central fixation due to amblyopia, in cases of false fixation, and also in children too young to cooperate. This last constitutes a very grave defect, as it is imperative to treat children early in order to get the best results. These two tests, which are allied, are the *phorometer* and the *screen tests*. Savage[7] has aptly described the phorometer test as follows:

"A red glass should be placed before the good eye. Before the deviating eye should be placed the rotary prism in the position for testing for lateral heterophoria, with the displacing prism of six degrees, base up, in the cell toward the eye. The red and the yellow blazes of the candle or gas jet having been found, the latter will appear more or less removed from the vertical line passing down through the red light and in the direction corresponding to the deviating eye. The fifteen-degree supernumerary prism should be placed, base out, in the front cell, which will carry the yellow light just that far toward the vertical line passing through the red light. Revolving the rotary prism in the nasal arc, the yellow light is carried still nearer the vertical line, which it may be made to read at some point between zero and ten degrees; but if this falls short, the rotary prism should be revolved back to zero, and a stronger supernumerary prism (twenty degrees, twenty-five degrees, or thirty degrees) should be placed in the anterior cell. Now the rotary prism should be turned again in the nasal arc and then stopped at that point where the patient declares the yellow light directly under the red one. If the twenty-five degree prism is in the front cell and the rotary prism stands at seven degrees, the prism degree measurement of the esotropia is thirty-two degrees, one-half of which would give the degrees of arc, viz., sixteen degrees. As already stated, the angle gamma does not have to be considered in connection with the phorometer measurement."

The screen test, which has been specially introduced and advocated by Duane[4] in this country, is described by him as follows:

"The patient is directed to look sharply at the test object, and then a card is placed before the right eye and passed quickly to and fro from one eye to the other.

"If there is *orthophoria,* neither eye will deviate when covered, and, consequently, will remain steady when the screen is removed. If, however, there is *squint* or *heterophoria* of more than a degree or two, each eye when covered will deviate, and when uncovered will turn back into the fixing position. Thus, if there is either an esophoria or convergent squint, each eye in turn will deviate in or toward the nose when covered, and will swing out again when uncovered.

"In squint, whether noncomitant (paralytic) or comitant, the deviation of the squinting eye behind the screen is called the primary deviation; that of the nonsquinting eye is the secondary deviation. In paralytic squint, the secondary deviation exceeds the primary (page 737); in comitant squint, usually, but not always, it equals it.

"The *amount* of the deviation can be roughly determined by inspection, but is best measured accurately by placing a prism before the eye (apex in for esophoria and convergent squint, apex out for exophoria, and divergent squint, apex down or up for hyperphoria) and increasing the strength of the prism until the screen-deviation is first abolished and then converted into a movement in the opposite direction. The first prism that causes this overcorrection, less two prism-degrees, represents the amount of the deviation."

In describing the various methods of determining the deviation of squint, Duane[4] says:

"No one of these methods at all approaches the screen test either in accuracy or in convenience. They do not show, or at least they show very imperfectly, the character of the squint (whether alternating or constant, periodic or continuous). Nor can they measure a vertical and a lateral deviation simultaneously, as the screen test does. Nor can they measure well, as it does, low degrees of squint, and they cannot measure heterophoria at all. Nor can they show, as it does, the varying amount of the deviation in different directions of the gaze."

These subjective tests are particularly adapted to the measurement of squint in older subjects and the brighter class of younger children, in whom diplopia can be produced but with the amblyopic squint, and with the stupid or unruly child, their use becomes difficult and at times impossible.

R. I. Lloyd[8] claimed to be able to measure squint more accurately than has been done with any of the methods previously used. His description of his method is as follows:

"The method of combining the outlining of the blind spots and examination of the macular area of the squinting eye has been gone over in the preceding section, but we can simply say here that if the blind spot of the squinting eye is located when the good eye is fixing, we have a fixed point to compare with the same blind spot when the squinting eye is fixing and the good eye is closed. The difference in position of the blind spot when the eye is squinting and the blind spot when the eye is not squinting must be the accurate measure of the deviation. Some will ask how you can tell whether the squinting eye centers accurately, and the answer is, that the position of the blind spot of the deviating eye with the good eye closed and the blind spot on the recording chart should coincide or be close together."

I can make no comment on this method further than to say that such wide discrepancies as given in his table of measurements with the campometer and the perimeter would lead one to doubt the accuracy of the one or the other.

It is advisable to use two methods of measuring squint. In those cases in which the above subjective measurements cannot be made, I use both the perimeter and the Holzer modification of the Priestly Smith tapes and find that they are good checks, one on the other.

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CHAPTER VIII

PARALYTIC STRABISMUS

Paralytic squint is a deviation of the visual axes of the eyes caused by loss of power of the muscle. This is usually due to a lesion of the nerve supplying the muscle. The lesion may be peripheral or central, and it may be partial or complete. If complete, it causes entire loss of the action of the muscle, and is called a paralysis. If only a partial loss of action is present, it is called paresis.

Paralytic squint is differentiated from comitant squint by

(1) Limitation of movements.
(2) False orientation.
(3) Diplopia.
(4) Positional head tilting.
(5) Vertigo.
(6) Age at occurrence.

Limitation of Movements.—In comitant squint there is no limitation of the movements in any of the fields of regard except in very pronounced cases of long standing. The deviated eye maintains its relative degree of deviation from the fixing eye in all fields of regard. In paralytic squint, there is *always* limitation of the motion of the paralyzed eye, and this is in the direction of the action of the muscle affected.[1]

The degree of deviation is constant and the same degree in the different fields of regard in comitant cases, whereas, in paralytic squint the deviation varies with the position of the object looked at. If it happens to be in the field of the action of the paralyzed muscle, it will be greater than it would be if it were in the field of the action of the sound muscle.

In paralytic squint, when the sound eye fixes and the affected eye squints, the deviation is called *primary deviation.* If the sound eye is covered and the paralytic eye is forced to focus on an object, the unaffected eye will be seen to deviate much more than the affected eye. This deviation of the sound eye under cover is called *secondary deviation.* This excessive deviation of the sound eye is due to an excess of effort sent to the paralytic eye to force it into certain positions. This excess stimulation is sent to both eyes, and causes the extra deviation of the sound eye. In comitant squint the primary and secondary deviations are usually equal.

False Orientation.—If a patient with a recent paralysis of an ocular muscle is made to close his sound eye and walk toward a given point, being guided by the paralytic eye, he will stagger toward the action of the paralyzed muscle. This is due to the excess nerve energy sent to the paralyzed muscle in an effort to adjust the eye to the object of regard, and this is so much in excess of that which is normally demanded that the patient is under the impression that the object is much farther to one or the other side than it really is. If, now, the eye is uncovered and the patient is asked to point to an object within reach, he will invariably point to one side, to the side of the action of the paralyzed muscle. This is called Von Graefe's reaching test.

Diplopia.—Diplopia, or the seeing of double images, is the most annoying symptom in ocular paralysis and, at the same time, of the greatest diagnostic importance.

Homonymous Images.—When the image of the paralyzed eye is on the side of the affected eye, the diplopia is called homonymous.

Heteronymous Images.—When the image of the paralyzed eye appears on the opposite side, i.e., on the side opposite to the affected eye, it is called heteronymous.

The restriction of motility, strabismus, and diplopia make their appearance when the eye is turned in the field of action of the affected muscle, and become more marked as the effort is made to turn the eye more and more to the affected side, and the converse is true; that is, the squint, loss of motility, and diplopia are lessened or disappear entirely as the eyes are turned in the opposite direction. Take, for example, a complete paralysis of the right external rectus. We have internal squint on looking straight ahead, and the amount of deviation and the diplopia both increase rapidly as an effort is made to look to the right; in fact, both diplopia and squint are present until the gaze is directed to the left of the median line some 15 to 20 degrees. The behavior of the double image in the various fields of regard is without doubt our best diagnostic aid in determining the muscle paralyzed and the degree of paralysis present.

In persons with a recent paralysis, this diplopia is particularly annoying, but by tilting the head, or shutting one eye, the habit of avoiding the diplopia is soon formed. In some cases of long standing the image of the deviated eye is practically suppressed, and thus the diplopia no longer annoys.

Head Tilting.—In order to avoid diplopia, which is not only annoying but sometimes dangerous, many individuals learn to tilt their heads in the direction of the action of the paralyzed muscle. By doing so, the images of the objects are made to blend, and they are thus rid of the diplopia. Take, for instance, the paralyzed right external

rectus. The action of that muscle is to turn the right eye out, i.e., to the right. In order to relieve that muscle from work, the patient turns his head to the right in order to relieve all action of the external rectus. In other words, in paralysis of the external rectus, the eye turns in, and the head is turned in the opposite direction to the position of the paralyzed eye in order to avoid the diplopia.

In paralysis of the right rotators, the patient turns his head to the right. In paralysis of the left rotators, the head is turned to the left. In paralysis of an elevator or depressor, he tilts his head upward or downward, and in addition he tilts his head toward one or the other of his shoulders, i.e., he holds his head in an oblique position. This is usually more pronounced than the elevation or depression. Duane[2] says:

"A vertical diplopia of 25° or more may be obviated in this way, the patient tipping the head toward the right or left shoulder according as the right-hand or left-hand image is higher. The patient may combine the side-tipping with more or less lateral rotation and up and down tipping, but the side tipping is the predominant, and often the only, abnormality of attitude."

Vertigo.—Vertigo in paralytic strabismus is due to the diplopia and to the use of the affected eye. The confusion of the double images in recent cases of paralytic squint often causes vertigo, but the most annoying vertigo is due to the use of the paralyzed eye. When the object observed lies in the opposite field to the action of the muscle, vertigo is not observed, but when it lies in the field of the action of the muscle, objects are located too far to that side, and the more so the farther the eye is turned to that side.

As the gaze passes from the field of regard in which the paralyzed muscle is not called upon to act to that field in which it should act, but can not on account of the paralysis, the objects appear to fly with increased velocity in the direction in which the eye is moving. It is this apparent movement of objects which causes the vertigo. This is so severe at times as to cause nausea. This is called visual vertigo, and is distinguished from aural vertigo by being relieved on covering the affected eye.

Age at Occurrence.—It is well known that comitant strabismus usually develops before the fifth or sixth year. A strabismus occurring after that age is usually paralytic.

Paralytic squint in children usually follows some serious constitutional disease such as diphtheria, syphilis, or infantile paralysis. In older subjects syphilis, focal infections, and cardiovascular diseases, associated with cerebral hemorrhages, are usually the cause.

One characteristic of ocular paralysis is that it is very annoying at first, due to the diplopia, and to disturbance in orientation associ-

ated with vertigo. These are lessened as the paralysis lessens; and even when the paralysis becomes permanent, in time the vertigo disappears and allowance is made for the false positions of objects, and after a long time more or less suppression of the false image takes place. While the vertigo and diplopia become less in complete paralysis of long standing, the strabismus is increased, due to overaction and contraction of the opposing muscle and no doubt to loss of tone and atrophy of the affected muscle. In many patients the deviation is as marked as seen in comitant strabismus, and thus may be confused with it. Duane was of the opinion that many cases of comitant squint had their origin in a paralysis or a paresis.

Paralysis of a single muscle is most frequently noted in the external rectus. This is due in the first place to the fact that the external rectus and the superior oblique are the only muscles which have individual nerve supply (the abducens and trochlear). A second reason for the external rectus being most frequently affected is that the sixth nerve is more exposed in its course than the other ocular nerves. The other ocular muscles are all supplied by the motor oculi communis; hence they are not so frequently affected singly. Duane[2] says:

"Isolated paralysis of the superior rectus is quite common (more frequent than trochlear paralysis), and pareses of the inferior rectus (usually very slight) are fairly common."

Paralysis of the oculomotor nerve causes a drooping of the upper lid, dilation and fixation of the pupil, paralysis of accommodation, deviation downward and outward of the eye with slight exophthalmus.

There are certain lesions which do not affect individual muscles but affect those muscles which produce associate movements, such as looking up or to right or left. Such a paralysis is termed *conjugate paralysis*. The cause of conjugate paralysis is due to lesion in the conjugate centers in the brain.

Diagnosis of Ocular Paralysis.—The diagnosis of ocular paralysis is made by observing, (1) the deviation present, (2) the loss of movement of the paralyzed eye, (3) double vision (diplopia), (4) vertigo and confusion in orientation.

Deviation.—The deviation is determined by covering first one eye and then the other and observing the position of the covered eye in the six diagnostic directions. It will be observed that the paralyzed eye when behind the screen will deviate, and this deviation will increase as the eyes are carried in the field of the action of the paralyzed muscle. The paralyzed eye will lag behind in the field of action of its paralyzed muscle, and the deviation will always be in an opposite direction to the normal action of that muscle, due to the unopposed

action of its antagonist. The deviation of the sound eye, when the affected one is being used, is always greater than that of the affected eye (secondary deviation).

The measurement of the deviation is best made by the use of that prism which abolishes the movement of redress. It is essential in making a study of a paralytic case that the head be kept steady and erect.

Loss of Movement.—The loss of movement is best determined by making the eyes follow a bright object in the six diagnostic directions and observing in which field the paralyzed eye lags. This always occurs when the object is brought into the field of action of the paralyzed muscle, and this lagging will become more and more pronounced the farther the object is carried in this field. This is known as the excursion test.

Diplopia.—E. and M. Landolt[3] give the following rules for detecting the paralyzed muscle by observing the double image produced by regarding a candle or vertical slit of light some twenty feet away, one eye being covered with a red glass:

1. To note the nature of the diplopia, *homonymous, crossed* (heteronymous), or *vertical.*

2. To determine the paralyzed eye by the law that "The affected eye is recognized by the fact that the diplopia increases when the direction of vision is turned towards its image."

3. To find the paralyzed muscle by the law that "The paralyzed muscle is the one which would have placed the eye in the position and direction of the false image."

The above rules suffice for finding the paralyzed muscle acting in the horizontal plane. It will be seen that it is much more difficult to locate the paralyzed muscle acting in the other fields of regard.

Banister[4] gives a very lucid and accurate chart for determining the paralyzed muscle. (Figs. 48 and 49.)

The diagnosis of ocular palsy and the determination of the muscle affected by means of the double images produced is probably best made by the use of the tangent plane or modified Bjerrum curtain. In this test the patient is seated 75 cm. in front of the screen, with head adjusted so that the center pin is directly in front of, and on a level with, the eyes. A red glass is placed before the right eye, and a small electric light is carried over the curtain in the six diagnostic directions, and the points where diplopia occurs are marked with pins. This maps out the field of single vision. In the regions where diplopia occurs, the patient sees two lights, one red, that of the right eye, and one white, the image of the left eye. A black pin is thrust into the curtain at the position of the white light and a white one in the position of the false or red light. The positions of the pins indicate rela-

DR. BANISTER'S MUSCLE CHARTS

ADDUCTOR PARALYSIS

Heteronymous Diplopia

RIGHT EYE AFFECTED

Diagnosis	In	Out	Muscular Actions
Paralysis of right superior rectus.			The superior rectus turns the cornea upwards and inwards and rotates the upper extremity of the vertical meridian inwards.
Paralysis of right internal rectus.			The internal rectus turns the cornea horizontally inwards.
Paralysis of right inferior rectus.			The inferior rectus turns the cornea downwards and inwards and rotates the upper extremity of the vertical meridian outwards.

Fig. 48.—Dr. Banister's muscle charts, adductor paralysis.

ABDUCTOR PARALYSIS

Homonymous Diplopia

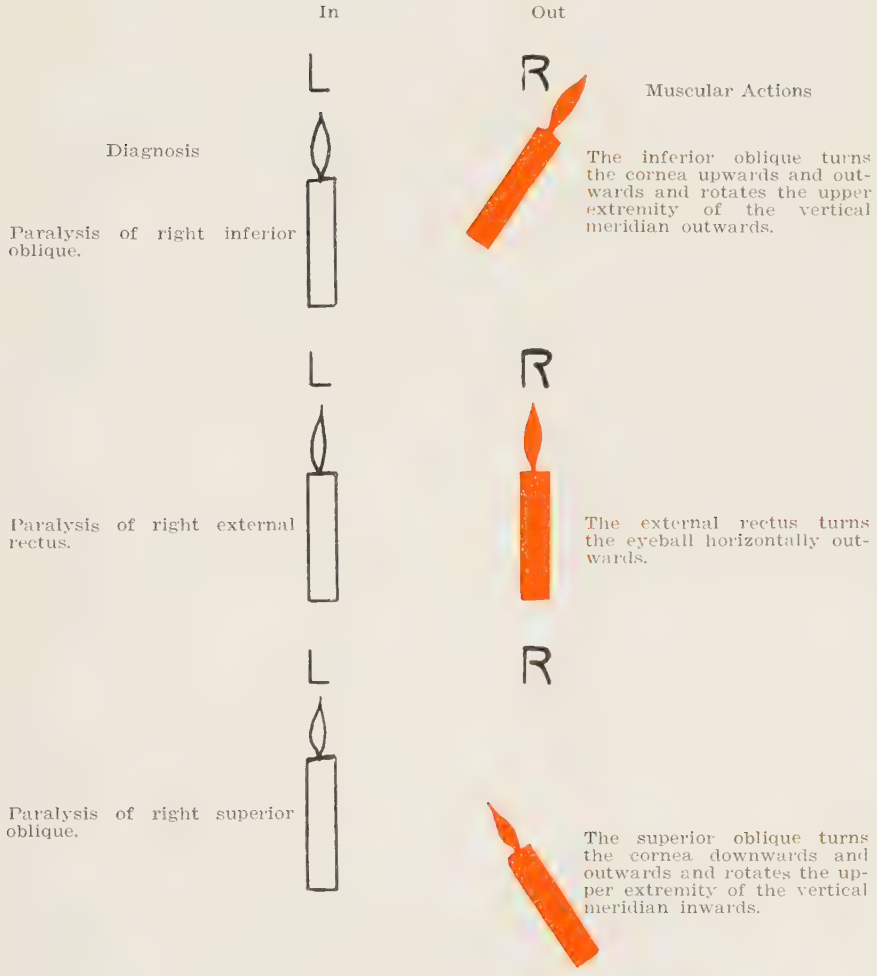
In
Out
L
R
Muscular Actions
Diagnosis
The inferior oblique turns the cornea upwards and outwards and rotates the upper extremity of the vertical meridian outwards.
Paralysis of right inferior oblique.
L
R
Paralysis of right external rectus.
The external rectus turns the eyeball horizontally outwards.
L
R
Paralysis of right superior oblique.
The superior oblique turns the cornea downwards and outwards and rotates the upper extremity of the vertical meridian inwards.

Fig. 49.—Dr. Banister's muscle charts, abductor paralysis.

N. B. The false image is always displaced in the direction of the normal action of the paralyzed muscle, and to a degree commensurate with the movements of the fixation object in that direction.

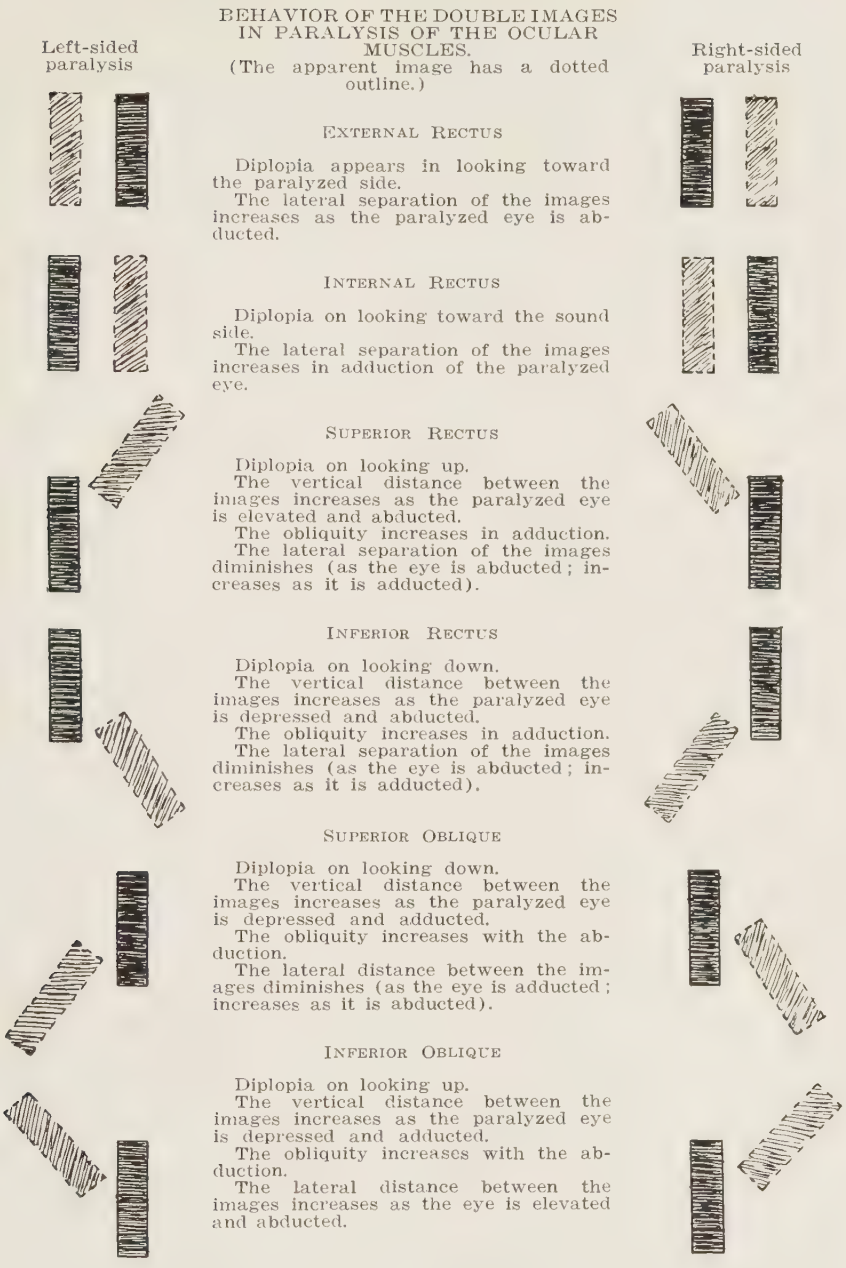
BEHAVIOR OF THE DOUBLE IMAGES IN PARALYSIS OF THE OCULAR MUSCLES.
(The apparent image has a dotted outline.)
Left-sided paralysis
Right-sided paralysis
External Rectus
Diplopia appears in looking toward the paralyzed side.
The lateral separation of the images increases as the paralyzed eye is abducted.
Internal Rectus
Diplopia on looking toward the sound side.
The lateral separation of the images increases in adduction of the paralyzed eye.
Superior Rectus
Diplopia on looking up.
The vertical distance between the images increases as the paralyzed eye is elevated and abducted.
The obliquity increases in adduction.
The lateral separation of the images diminishes (as the eye is abducted; increases as it is adducted).
Inferior Rectus
Diplopia on looking down.
The vertical distance between the images increases as the paralyzed eye is depressed and abducted.
The obliquity increases in adduction.
The lateral separation of the images diminishes (as the eye is abducted; increases as it is adducted).
Superior Oblique
Diplopia on looking down.
The vertical distance between the images increases as the paralyzed eye is depressed and adducted.
The obliquity increases with the abduction.
The lateral distance between the images diminishes (as the eye is adducted; increases as it is abducted).
Inferior Oblique
Diplopia on looking up.
The vertical distance between the images increases as the paralyzed eye is depressed and adducted.
The obliquity increases with the abduction.
The lateral distance between the images increases as the eye is elevated and abducted.

Fig. 50.—Behavior of the double images in paralysis of the ocular muscles. (Fuchs' *Textbook of Ophthalmology.*)

tions of the double images, and their degree of separations in the different fields of regard aids in determining the muscle involved. It is always best, where the patient will fix with either eye, to make these tests with the red glass first on one eye and then on the other, as the deviations will become more marked as the affected eye is

being used, due to the secondary deviation being always greater. Duane[2] gives us an excellent chart of the behavior of the double images in ocular paralysis (Fig. 50).

It must be remembered that the image formed by the paralyzed eye always lies on the side toward which the diplopia increases, and that the image belonging to the paralyzed eye is displaced in the same direction in which the affected muscle, if acting, would move the eye.

Tilting of the Double Image.—Since tilting of the eye is affected by two sets of muscles, the two obliques and the superior and inferior recti, it becomes necessary to consider the actions of these muscles to make a diagnosis by the tilting of the images. The superior rectus and superior oblique intort the eye; hence the meridian of the eye when these muscles are affected will be extorted, or tilted out, and the image of objects seen will be tilted inward If the superior rectus is affected, the image will be above. If it is the superior oblique, the image will be below.

The inferior rectus and inferior oblique rotate the vertical meridian outward and in paralysis of these muscles the vertical meridian is turned in, or intorted, and the image formed will be tilted outward. If the inferior rectus is affected, the image is below, and if inferior oblique it is above.

Difficult Cases.—In cases of paresis, and in those with several muscles paralyzed, it becomes increasingly difficult to locate the affected muscle or muscles. Unequal visual acuity may cause the patient to use the paralyzed eye, also previous muscular imbalance; that is, an esophoria or exophoria might add to our difficulty, as well as contractures from old paralyses. A want of intelligence on the part of the patient, or at times a want of cooperation on the part of the patient, adds to our difficulties.

Location of the Lesion.—Ocular palsies are due to some lesion either in the cortical centers or the nerve tracts from them to the eye. These lesions are divided into intracranial and orbital. Intracranial lesions are those which are located in the cranial cavity. These various lesions are named according to the location of the lesion. These are best divided into *supranuclear* and *nuclear*.

Supranuclear lesions are those affecting the cortical centers (cortical paralysis), the vestibulocerebellar tracts, or the conducting paths from the above two to the nerve nuclei located on the floor of the fourth ventricle. A lesion of the fibers connecting these centers is called *fascicular* or *radicular* paralysis. Where the nerve trunks are affected along the base of the skull it is termed *basal* paralysis. Supranuclear lesions never cause conjugate paralysis, or paralysis of convergence or accommodation, but at times they cause ptosis.

Nuclear Lesions.—These comprise injury or disease affecting the nuclei of the ocular nerves. They usually cause paralysis of several ocular muscles (central ophthalmoplegia), and these paralyses are usually bilateral; inasmuch as fibers go from each oculomotor nucleus to both eyes, any extensive lesion would affect both eyes. Extensive lesions of the nuclei cause paralysis of all the ocular muscles (total ophthalmoplegia), both exterior and interior. Slight nuclear lesions may affect only one or two muscles, or may cause paralysis of external ocular muscle only (ophthalmoplegia externa), or may affect the pupil and accommodation only (ophthalmoplegia interna). This latter is frequently found in patients suffering with cerebral lues, and is also found in early stages of tabes and of paresis (which are often luetic). Acute cases occur from various poisons.

Fascicular Paralysis is diagnosed when there is a paralysis of an oculomotor nerve on one side and at the same time a paralysis of the extremities of the opposite side.

Basal Paralysis is indicated in (a) unilateral total paralysis, unilateral total oculomotor paralysis, or when a number of the cerebral nerves of that side of the face (facial, auditory, optic, and olfactory) are paralyzed one after the other. (b) When the affection of the trigeminus begins as a neuralgia in central paralysis. (c) Disturbances of vision, unilateral blindness without ophthalmoscopic findings, also temporal hemianopsia.

Orbital Paralysis.—When the paralysis is due to injury to the nerve or muscles within the orbital cavity, it is called orbital paralysis. The diagnosis of orbital paralysis is usually made by the accompanying symptoms which indicate some lesion within the orbit. These are pain in the orbit, tenderness on pressure, evidence of tumor in orbit, which may be detected by palpation or by an exophthalmus, unilateral optic neuritis, or evidence of trauma. The most frequent causes of orbital paralysis are tumors, injuries, inflammations of the orbital tissue, the latter due in large measure to infections of the accessory nasal sinuses. Other causes can at times be found for ocular pareses which are transient in their nature. These are considered to be due to colds, and are spoken of as rheumatic paralyses. This condition is usually a true neuritis. Some few cases are myogenous, due to either a degeneration, inflammation, or nondevelopment of the muscle. These probably best explain Treacher Collins' hereditary ophthalmoplegia externa. Myasthenia at times affects the ocular muscles, causing a paresis which is increased rapidly by exhaustion.

Congenital Paralysis.—This probably occurs more frequently than the average textbook would indicate. The most frequent muscle to be affected is the superior rectus. These lesions are often incomplete and are readily overlooked. Slight paresis of the inferior oblique is

fairly common. In congenital paralyses of the external and internal recti, we get what is known as *retraction syndrome;* when the eye is directed inward it retracts into the orbit and deviates upward, at the same time the palpebral fissure closes. Many cases of congenital paralysis are due to either weak, or absent, muscles, or to birth injury.

Prognosis.—In ocular palsies the prognosis must always be guarded. The first indication is to find the causes, and these are not always apparent even after the most exhaustive study. The duration of the paralysis and the accompanying symptoms must be considered in making our prognosis. The longer the paralysis has been present, the graver the prognosis.

Treatment of Ocular Palsies.—Of necessity, the first indication is to find the cause. A thorough serologic and physical examination of the patient is essential. Syphilis, sinusitis, rheumatism, orbital tumors, and cerebral lesions must all be investigated, and by competent assistants. Where any of the above are demonstrated, they must receive proper treatment. In syphilis, the arsenical preparations are indicated, followed by mercury, and at times massive doses of iodides. If the sinuses are infected, energetic local or operative treatment must be conducted by a competent rhinologist. In rheumatic cases, after eliminating all foci of infection, alkalies and salicylates with proper dietetic regimen are indicated. In chronic cases electricity is often of benefit.

Operative Treatment of Paralytic Squint.—Operative procedures are not justifiable in any recent paralytic squint. Usually it is advisable to wait one year after the most exhaustive treatment has failed to give relief before an operation is advisable. In congenital squint, associated with head-tilting and other evidence of discomfort, we should operate early. As in comitant squint, there are two types of operative procedures which can be followed. One is to weaken the strong or overacting muscle by tenotomy or recession, and the other is to strengthen the action of the weak muscle by shortening or advancing its insertion. This is done by advancement, tucking, or resection operations. (See chapter on Operations.)

The correction of a paralytic squint due to a lesion of one of the lateral muscles is best done by means of an advancement or resection of the paralyzed muscle accompanied, at the same time, by a tenotomy or recession of the overacting antagonist.

In vertical paralysis, the paralyzed muscle should be shortened and if necessary a partial tenotomy or a recession done on the antagonist of the same eye. When this work is measured, very satisfactory results can be secured.

Duane[2] advises the tenotomy of the inferior oblique of the sound eye in paralysis of the superior rectus. Posey and others have advocated this procedure. It is efficacious in congenital cases in which there is a marked upshoot of the sound eye when the paralytic eye fixes. I must agree with Banister, however, that there is serious objection to sacrificing the function of the inferior oblique if the eye can be straightened otherwise.

The operative indications in paralyses of the obliques are well stated and illustrated by Banister[4] as follows:

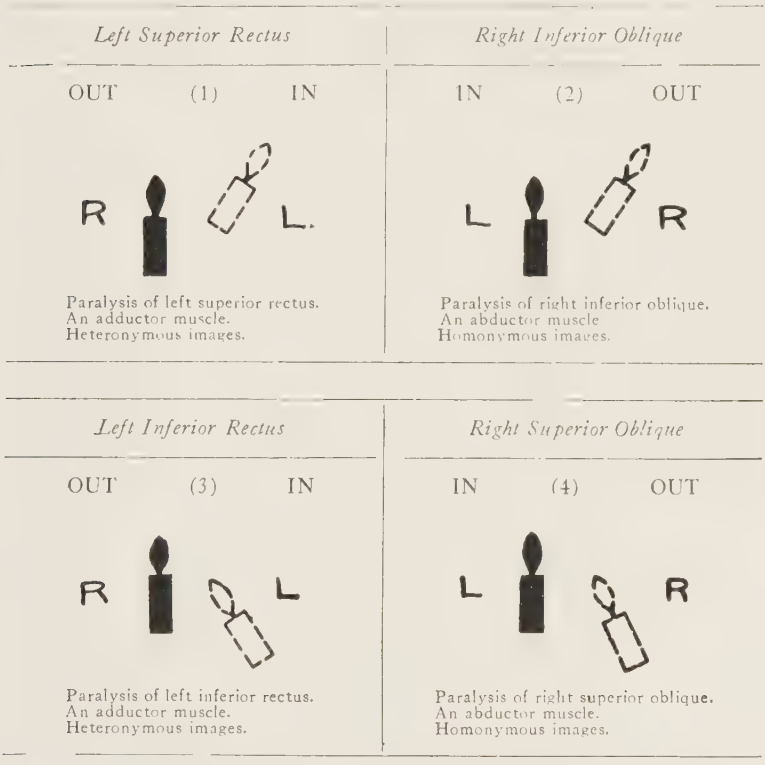

Fig. 51.—Dr. Banister's diagram for paralysis of the obliques with operative indications on the contralateral muscles.

"In paralysis, on the other hand, of either the *superior* or *inferior* oblique, this limiting of the operation to the affected eye is impracticable from anatomical considerations, and the respective contralateral muscles must be set back by tenotomy to secure relief from the diplopia, by inducing a deviation of a similar character in the case of the sound eye. Fusion then becomes attainable. The principles involved will be illustrated in the accompanying diagram, which the writer has devised. (For positions assumed by the images of the paralyzed eyes see Fig. 51.)

"Similar states of contralateral parallelism would occur in simultaneous paralyses of the right superior rectus and left inferior oblique, on the one hand, and of the right inferior rectus and the left superior oblique, on the other. In consulting the diagram just referred to, it can be seen, for example, that if the right inferior oblique muscle is paralyzed, the image of the right eye will assume the position of R (2). If the contralateral left superior rectus is purposely set back by a tenotomy, it will lose power over the globe sufficiently to cause the image of the left eye to take a position approximating that of L (1). The images of the two eyes, R (2) and L (1) will therefore harmonize in position, and can thus be fused with relief of the diplopia. This diagram will apply with equal pertinence to other contralateral problems."

References

[1]Landolt: Archiv. d'Ophth. et du Strabisme, 1887, xvii, 74.
[2]Duane: Fuchs's Textbook of Ophthalmology, ed. 6.
[3]Landolt, E., and M.: Defective Ocular Movements and Their Diagnosis, London, 1913, p. 51.
[4]Banister: The Extrinsic Muscles in Health and in Disease, American Optical Co., 1924, p. 78; p. 82.

CHAPTER IX

THE EXAMINATION OF A CASE OF STRABISMUS

An exhaustive and systematic examination is absolutely necessary in every case of squint. This is particularly true in young subjects in whom it may yet be possible to secure binocular vision. By this means only is it possible to obtain the necessary facts for laying out a rational and scientific course of treatment to be carried out in a given case. We are indebted to Claude Worth for his insistence upon the early and scientific analyses of cases of squint. The following method of examination of squint cases is slightly modified from that recommended by Worth:[1]

(1) History.
(2) The type of the squint.
(3) The power of fixation in the deviating eye.
(4) Movements of each eye.
(5) Convergence and accommodation.
(6) Determination of the visual acuity.
(7) The determination of the degree of binocular vision or fusion.
(8) The degree of deviation.
(9) The refraction.
(10) Ophthalmoscopic examination.

The above methodical examination I have used for years. At a glance, it seems superfluous; however, I feel that every step is necessary if we are to know all that we should know before proceeding radically with the treatment of a case of squint.

History.—(a) Age at onset, and duration. This is most important, and should be obtained with as much accuracy as possible. It often gives us an insight into the nature of the squint, and aids in directing the treatment. If the deviation is from birth, the possibility of its being a congenital squint (associated with more or less amblyopia due to a defect in one or both eyes, such as scars on the cornea, congenital cataract, or some central fundus lesion) should be considered. If the squint occurs between the second and third years, and is monocular, amblyopia is apt to occur early and become marked if means are not used for its relief.

Duration.—If the case is of only short duration and periodic, it will in all probability be relieved by the use of glasses and orthoptic exercises. If it is of long standing, with a marked degree of blindness, monolateral, and of high degree, glasses and exercises will probably be of little avail, and some operative procedure will be necessary.

(b) The second thing to be considered is the mode of onset — whether intermittent, alternating, or constant, and the duration in each class. This also is of diagnostic importance, as a case which is intermittent is more apt to yield to treatment than one which is practically constant from the start.

(c) *Supposed Cause.*—The parent invariably has some specific idea as to what caused the child's eyes to cross. It is always of interest, and is often instructive, to obtain these ideas. There is no question but that many of the acute illnesses are contributing causes of squint, as has been mentioned elsewhere.

(d) *Heredity.*—Donders claimed that he found heredity to have very little or no influence in squint cases. Browne and Stevenson thought that there was quite a hereditary tendency. Worth has shown us there is a decided relation to heredity in squint. I have found that those cases with hereditary histories require earlier operative interference and as a rule a longer fusion training to secure binocular vision than is required where the element of heredity is not present. It is in this particular type of case that we are apt to find essential squint, or, as Graefe first described this condition, "antipathy to binocular vision." Worth found 51.78 per cent with more or less remote hereditary connection. There are, however, many cases in which for generations there is no relative, even remote, who has been affected with squint.

The Type or Character of Squint.—(See chapter on Types of Squint). Worth classifies squint into:

(1) Occasional squint, of which there are two classes: (a) Premonitory occasional; (b) true occasional.

(2) Constant unilateral squint.

(3) Alternating squint, of which there are two distinct classes: (a) Accidentally alternating squint; (b) essentially alternating squint.

Savage[2] describes esotropia under periodic, alternating, and permanent, stating that the same case at different times may present all these conditions.

Duane,[3] who has given this subject as much thought as any one in America, divides squint into the following:

"1. *Constant,* if present all the time; *intermittent,* if, under the same conditions of vision, it is sometimes present, sometimes not. (This is the periodic of most writers.)

"2. *Continuous,* if present to about an equal degree in both distant and near vision; *periodic,* if very much greater for near objects than for distance, or vice versa. (In former case it is said to be directly, in the latter, inversely periodic.)

"3. *Comitant* (or concomitant) if the amount of deviation remains the same, and *noncomitant* if it changes notably, as the eyes are carried in different directions.

"Finally, a squint is called monocular, if with both eyes open, it always affects the same eye, and is then called right or left, according to the eye affected; and it is called alternating if, with both eyes open, sometimes one eye, sometimes the other, deviates."

It is exceedingly important to note the type of squint present and its duration. This aids materially in our prognosis as to time of treatment, nature of same, and duration, and the ultimate end-results to be expected. In periodic intermittent cases of only a short duration, one would expect an almost immediate relief of the condition, after a careful refraction and adjustment of the proper glasses and orthoptic training as indicated in the chapter on Nonoperative Treatment of Strabismus.

If it is of an alternating type, has been present since two years of age, or earlier, and of marked degree, glasses will probably not relieve it, and an operation will in all probability be a necessity. If it is constant, and of but short duration, with good acuity of vision and of low degree, glasses, atropin, and exercise will probably bring about a cure. If constant, of long duration, high degree, marked amblyopia, glasses and atropin will probably not cure, and an early operation may be advisable. Thus we see the immediate and remote importance of determining at first the character of squint present.

Simple inspection will usually reveal the type of squint to the experienced oculist; however, a case with high angle gamma or the overhanging of tissue in the inner angle may cause the appearance to mislead us and further examination may become necessary. This is best determined by the monocular fixation test, binocular fixation test, or, best of all, the mirror test.

Monocular Fixation Test.—We cover one eye with a card in such a manner that the eye can be observed and at the same time request the patient to fix attentively some distant object. An observation of the uncovered eye will inform us if it is fixing centrally or eccentrically, or not fixing at all. Any marked deviation of the covered eye will be apparent.

Binocular Fixation Test.—Inspection of the positions of each eye when directed at some distant object will usually indicate whether there is any marked deviation. By means of the screen test, i.e., alternately covering one eye and then the other with a card, we are enabled to detect even the slightest degree of deviation.

Where there is orthophoria, neither eye will deviate when covered; if, however, there is some heterophoria or heterotropia present, the eye will deviate when covered. In case of convergent squint, when the one eye fixes the other will turn in, and will straighten when uncovered and the other screened. The deviation of the eye behind the screen

is called the *primary* deviation. The amount of the deviation present can best be measured in intelligent patients and those old enough to cooperate by placing prisms in front of the deviating eye of such strength as to abolish the deviation on being covered and uncovered. Duane advises placing prisms in front of the eye until the eye is caused to deviate in the opposite direction, and "the first prism that causes this overcorrection, less 2 prism degrees, represents the amount of the deviation."

Mirror Test.—This is a most satisfactory way of determining whether or not strabismus is present. The patient is seated in a dark room with a light behind him. The surgeon is seated about two feet in front, and reflects the light into the patient's eye by means of the mirror of the retinoscope or ophthalmoscope. The patient is told to look at the mirror, when a tiny, bright image of the light is seen at or near the center of the cornea. The light is now flashed first on one eye and then on the other. Any deviation is at once detected by the position of the image on the cornea. In comitant squint the primary and secondary deviations are usually equal. In paralytic squint the secondary deviation is greater, and in spastic squint the primary deviation is usually greater.

Fixation of the Deviating Eye.—It is important to know whether the deviating eye fixes properly. This simple test is best made in the dark. The patient is seated with a light behind him, and is asked to look at the retinoscopic mirror as the light is reflected into his fixing eye. The position of the image is noted on the cornea. Now the fixing eye is covered and the light is flashed in the previously deviating eye. If it possesses central fixation, this eye will immediately become straight and the image of the light will be in the same relative position on the cornea as it was on the previously fixing eye. If there is an eccentric fixation, the eye will fix the light with this spurious macula, but a certain amount of deviation will still be present. This test is important, as cases with no fixation indicate marked amblyopia. These cases, and those with false fixation, are more difficult to cure than those in which central fixation is well developed.

Movements of Each Eye.—This test is made by covering one eye and requiring the patient to look into the six diagnostic fields of regard. This can be done with young children by using a flash light in a dark room. Adversion until the cornea is within one or two mm. of the caruncle and abversion of the cornea to the external canthus should be considered normal. Any marked want of deviation in the other cardinal directions can be readily detected. This test enables us to detect an overaction or underaction of any set of muscles. The muscles which are usually most affected in squint cases are the

lateral rotators. Often the interni overact and contractures take place, while the externi may atrophy or become markedly insufficient. The tropometer of Stevens is of great aid in determining the muscular status, and should be used in every case where possible (Fig. 52). In very young subjects accurate measurements are often difficult or impossible to make, but with patience and repeated efforts much can be done even in the youngest. This simple procedure is too often neglected. It aids very materially in determining an overacting internus or an insufficient externus. There is not usually any marked overaction of the interi in recent cases of squint, but in old cases it often becomes marked, and a knowledge of this is of much benefit in determining what operative procedure to use.

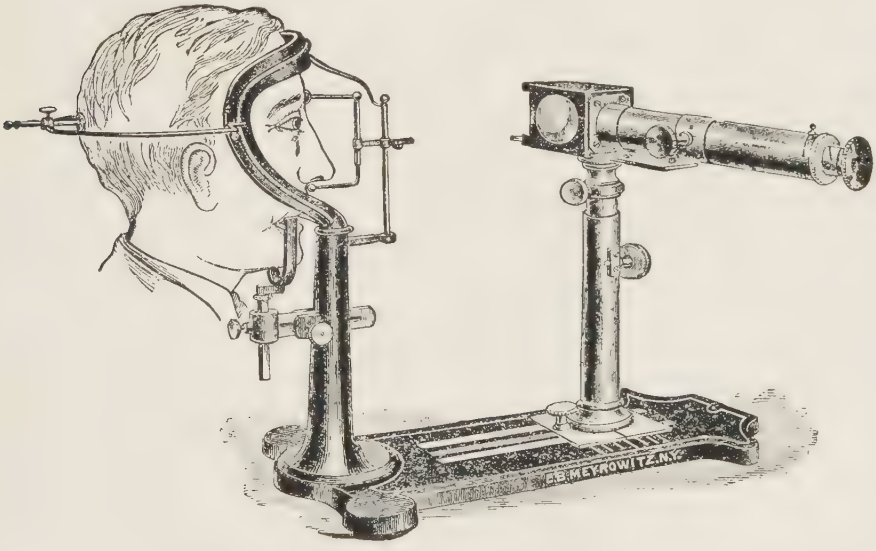

Fig. 52.—Stevens' tropometer.

Convergence.—It is advantageous to note the static and dynamic convergence in squint cases. In persons with orthophoria, looking at a distance there is no convergence, but if he has convergent squint, there is a static convergence equal to the angle of the squint. If he now looks at a near object, a certain amount of dynamic convergence is required in addition to his static convergence. This added convergence causes the already deviating eye to turn in more, hence the degree of squint is usually greater for near than for distance. This is often spoken of as accommodative squint, inasmuch as many patients are enabled to control their static squint until the dynamic convergence is called into action by the eye accommodating to see a near object. The near point in cases of squint can be measured by having the straight eye fix an object which is being gradually brought toward

the eye, at the same time watching the movements of the deviated eye. As the object approaches the eyes, the deviating eye will be seen to partially correct itself. This point is the p. p. If this is very close to the eye, it indicates an excessive dynamic convergence. Duane[4] claims that practically all cases of comitant squint are produced by an "anomaly of convergence combined with one of divergence." He states that "The ordinary form of esotropia is due to a convergence-excess combined with a divergence-insufficiency." Cases in which the degree of deviation is markedly increased for near (periodic-Duane) and decreased at a distance are certainly due to an anomaly of convergence or divergence, or both.

Determination of the Visual Acuity.—This is one of the most important tests to be made in our examination. The acuity of vision is one of our chief prognostic guides. The greater the degree of acuity, the more hopeful; whereas, the greater the defect, the greater is our difficulty. Buffon first, and Javal afterwards, called our attention to the importance of improving the visual acuity as a means of curing strabismus.

The ordinary method of determining the acuity of vision is by the use of the Snellen test-types for distance. The type recommended by E. Jackson, and that by Ewing and Green, are efficient and accurate and we recommend their use. If the types are well selected, this method is efficient where the subject can read the letters. In cases of illiterates and in younger subjects, various illiterate charts may be brought into service.

Worth's ivory balls are very good for determining the vision in the very young children. These balls are of different sizes and are thrown on the floor one at a time at some distance from the child, who has one eye bandaged or occluded, and he is required to run and pick up the ball. His ability to go directly to the ball when placed at a certain distance enables the examiner, considering the size of the ball used, to arrive at a fair idea of his visual acuity. It is always well to conduct these examinations in the presence of the parents, as any marked amblyopia can be thus shown and their cooperation in the future more certainly secured.

The first step in the treatment of a case of squint is to bring up the visual acuity where it is found defective, and by means of accurately determining the visual acuity on the first visit, and subsequently, the progress of the case can be at all times determined. No case with marked amblyopia will ever develop very useful binocular vision or fusion, and no case of strabismus is ever really cured without fusion; hence, the importance of the visual acuity.

The Determination of the Degree of Binocular Vision or Fusion.—On looking at a distant object, an image of the object is formed on

each retina, but the individual is not aware of this fact. The two images fall on the two foveae and are converted into one image, and we see only one object. This combining of the two images into one mental picture is called *fusion*, or binocular vision. This is supposed to be controlled by a special center in the brain known as the fusion center, and the mental faculty by which fusion is effected is termed the *fusion faculty*.

Inasmuch as it has been long known that binocular vision or fusion was an essential to straight eyes, it becomes necessary in the study of squint to determine the degree of fusion present. We are greatly indebted to Javal, E. Landolt, Remy, Parinaud, and particularly to C. Worth, for their work in stressing the importance of binocular vision or fusion as an essential in the treatment of squint. No one can place an eye straight by any method, and keep it straight, without binocular vision or fusion. C. Worth[1] says:

"When the fusion faculty is fairly well developed, neither hypermetropia, nor anisometropia, nor heterophoria can cause squint."

To determine the degree of fusion, it is best to use some form of stereoscope. The amblyoscope of C. Worth as modified by Black is one of the best. The Holmes stereoscope, which is a modification of the Brewster instrument, is in common use in America. One of the best instruments is the Phoro-Optometer stereoscope of Wells.[5] Worth's classification of fusion into different grades is convenient for study, but it must be remembered that there are, as Linn Emerson remarked, "57 varieties of fusion." Worth divides fusion into:

First grade. Simultaneous macular perception.
Second grade. True fusion with some amplitude.
Third grade. Sense of perspective.

The use of colored glasses before the eyes, one red and the other green, to determine the presence of binocular vision, was presented to the medical congress at Geneva in 1880 by Javal. This was further perfected by M. Bravais, of Lyon, and by M. Donders, of Holland. Snellen colored glasses, known as the "Dot Test," is a very rapid and efficient method of determining the second grade of fusion. (See Fig. 53, "four dots test" in Chapter X, Nonoperative Treatment of Strabismus.) The use of the Hering drop test for the sense of perspective is one of the oldest and best. I have found the use of the amblyoscope with the various charts efficient for making ordinary tests for fusion. (See Chapter X on Nonoperative Treatment.) The determination of the degree of fusion present in each case is of great importance, as it is of the highest prognostic value. A squint of recent occurrence in a patient with good acuity of vision in each eye

with fairly good fusion can certainly be cured; whereas, if it be a case with poor fusion, poor vision, and of long standing, the prognosis is not so promising.

Measurement of the Deviation.—This is considered of so much importance that a chapter has been devoted to the subject. This should be accurately done on the first examination and on subsequent examinations until a very definite measurement has been secured, and this should be repeated once or twice a month thereafter until the patient is cured either with or without an operation. Our records of the progress or want of progress in these cases is our chief guide as to when to operate or on whom to operate. As to the methods for determining the angle of the deviation, I beg to refer the reader to the chapter on this subject.

The Refraction.—The refraction of the eye is determined by retinoscopy after the use of a 1 per cent solution of atropin. In very young children it is advisable to use an ointment or a weaker solution, as distressing symptoms have been provoked by the use of 1 per cent atropin solution in very young subjects. The importance of accurate, painstaking retinoscopy cannot be too strongly urged. It often requires much tact and at times a great deal of patience on the part of the surgeon to do a satisfactory retinoscopy on a very young child. This is so important, however, that it should be obtained even though it require several sittings. The importance of this can readily be seen, as this is the sole method of determining the refraction of these young subjects, and our end-results depend upon our accuracy. In case of defective fixation, the retinoscopy is best made by causing the fixing eye to look at an object held in such a position as to bring the deviating eye straight and then proceed with the retinoscopy on that eye. In young subjects in particular, it is advisable to determine the weaker refractive meridian of the eye with a sphere and use a cylinder to correct the opposite meridian. By this method one can better arrive at the proper axis for the cylindrical correction.

Where practicable, the vision should be tested with the correction placed before the patient's eyes. After the retinoscopy has been carefully checked and vision carefully and accurately recorded with correction, glasses should be prescribed and worn before the eyes recover from the atropin. It is my custom to order the full amount of correction of the hypermetropia less .50 D as recommended by Worth with full correction of the astigmatisms. In subjects older than five years of age, in whom it is noted that the eyes occasionally turn in with the ordinary distance lenses, on prolonged close application for near, I have been in the habit of prescribing, according to the method of Javal, an overcorrection of from 1 to 2 D for these children to use

Wilkinson's Strabismus Chart

Date

NAME ADDRESS PARENT

HISTORY	Age	Age of Onset	Duration	Mode of Onset	Heredity	Given Cause

TYPE of SQUINT	Direction	Constant	Intermittent	Monolateral	Alternating	Eye Crossed

FIXATION

R

L

EYE MOVEMENTS (Tropometric)

Date					Date					Date				
	R	L	Up	Down		R	L	Up	Down		R	L	Up	Down
R					R					R				
L					L					L				

CONVERGENCE

PP	Convergence Excess	Divergence Excess
	Convergence Insuff.	Divergence Insuff.

DEGREE of DEVIATION

	Date			Date			Date			After T. or Op.
	Lateral		Vert-	Lateral		Vert-	Lateral		Vert-	
	Near	Dist	ical	Near	Dist	ical	Near	Dist	ical	
R										
L										

Obverse side.

DEGREE of FUSION

Grade	Date
1st	
2nd	
3rd	

REFRACTION and VISUAL ACUITY

OPHTHALMOSCOPIC FINDINGS

REMARKS

Reverse side.

in studying, and find that many of them will maintain binocular vision in this manner, whereas they are unable to do so with their distance lenses. I have been using bifocals for this purpose in those patients in whom the eyes are straight for distance and squint on near use, and find them of much aid in some cases.

Ophthalmoscopic Study.—During our study of refraction, while the eyes are under the influence of atropin, it is necessary to examine the eye carefully with the ophthalmoscope. While it is true that the retinoscope will show us defects of the cornea and opacities in the lens when present, it is always desirable to make a careful study of the fundus at this stage. It is particularly desirable to know the state of the macular region and the condition of the optic nerve, and this information can be obtained by the use of a little tact, even in the youngest of children.

I devised the accompanying chart which I have found a great help in making a methodical examination, providing at the same time a systematic record which is of inestimable value in the direction of the treatment in each case.

References

[1]Worth, C.: Squint, ed. 5, Philadelphia, 1921, P. Blackeston's Son & Co., p. 78.
[2]Savage, G. C.: Ophthalmic Myology, Nashville, 1902, p. 430.
[3]Duane, A.: Fuchs's Textbook of Ophthalmology, ed. 8, Philadelphia, 1924, p. 270.
[4]Duane, A.: Fuchs's Textbook of Ophthalmology, ed. 8, Philadelphia, 1924, p. 323.
[5]Wells: The Stereoscope in Ophthalmology, Globe Optical Co., 1918.

TREATMENT OF STRABISMUS

It has been deemed advisable to divide the treatment of squint into two chapters.

The first chapter will deal with the *Nonoperative Treatment*, and will cover that subject rather fully, as I feel that with the early cooperation of the parents and the family physician, much more can be done than is being accomplished at the present time. Early scientific care of these young subjects will prevent both the amblyopia and the necessity for operative procedure later.

The second chapter will deal with the *Operative Treatment*. In this chapter are presented many operative procedures, all of which have sufficient merit to justify their presentation. In addition I have presented new and more definite procedures, with recommendations for earlier and more exact surgery.

CHAPTER X

NONOPERATIVE TREATMENT OF STRABISMUS

The first and most important step in the nonoperative treatment of squint is educational. Every mother ought to know that squint in her child is amenable to early treatment, and that it is *not* a simple thing that the child will *outgrow*, but a very serious matter if neglected. She ought further to know that the early treatment may be a very simple matter, whereas, if the child is neglected, the treatment of necessity becomes a more serious and tedious matter. Glasses, the use of atropin, and exercise will usually cure these patients when seen early, but when neglected, and vicious habits are formed, it takes years of most persistent exercise and treatment to secure the same result that could have been secured with ease, had the case been treated from its incipiency.

Javal[1] says:

"The reestablishment of binocular vision by exercise usually requires as long a time as that which has elapsed since the commencement of deviation. A child of eight who commenced to squint at four may be cured at twelve years of age."

Many, if not most, of the neglected cases can never be cured except by operating, and then they are never so well off as those who receive the proper early care and are cured without operating.

The mother and the public in general must be taught that the surgical operation for the relief of squint is *not* the simple matter of "snipping a little muscle," but is a most difficult and exact surgical operation. Every teacher in the graded schools ought to be taught that any cross-eyed child in her school is a neglected child, and should insist on its being relieved of its deformity.

The education of the mother and the teacher is not our biggest educational task; the educational work must begin at home, right in our own medical family. The *doctor needs* to know more about the squint case. He *needs* to *know* that the time to do the most good is from the very incipiency of the case.

Browne and Stevenson[2] say:

"It is unwise to prophesy, but I venture to say that, if general practitioners will recognize the fact that all children are liable to squint, and that squints in the periodic stage are curable, whereas those in the habitual stage are very difficult to cure, and that treatment should

be begun, no matter how young the patient may be, immediately after the first 'glide of the eye' has been observed, squint will almost vanish."

Browne and Stevenson here voice the opinion of most of the progressive ophthalmologists in this country, and I wish to add that if the oculist is ever to see these cases early enough, it will be at the advice and insistent counsel of the family physician and the pediatrist.

Mr. B. Harman[3] found that over 80 per cent were seven years of age or over when they first presented themselves for treatment. He also found of the monolateral squinters that 45 per cent had no, or dubious, fixation. He remarked that "treatment of such cases is little more than beauty doctoring."

Dr. D. W. Wells[4] says:

"Of all the private cases of esotropia seen by the writer during the last five years, only 25 per cent were less than six years old."

C. Worth tells us that after the age of six, or at most, seven years, the training of the fusion faculty is scarcely worth the effort, hence something must be done to awaken the physicians to their duty of referring these children for treatment earlier.

They must be taught that squint *is curable* in its incipiency, and they must also be informed that it is difficult and often impossible to cure neglected cases. They must know also that neglected cases cannot be cured by a *simple* operation, and that the mere fact that we get a cosmetic result *does not mean a cure at all,* but simply a relief of a deformity, with the loss of an eye. They should know that to advise a mother to "let the child alone; he will outgrow it," means not only a lost eye as far as a useful-functioning organ is concerned, but it usually means an expensive, tedious operation. I can assure you this is the penalty that these parents suffer for following such ill advice. No physician worthy of the name would give such advice if he were better informed, and it is the duty of the oculist to teach his fellow practitioner these facts.

Our third educational program is still closer at home; it is in our own ranks. Many, I fear I should say the majority, of the men who make a specialty of ophthalmology are not alive to this question. Many are even slower to operate. They hesitate to assume the responsibility of the cases in a definite way, and squint cases are the most neglected cases in ophthalmology. This should not be so. If an oculist does not wish to take the time, the trouble, the care, and the responsibility of these cases, he should send them to an ophthalmologist who will. He should be ashamed to neglect these cases under his very eye. I regret to say that I see it almost daily, and it must be fairly general. Many cases are fitted, refitted, and fitted again,

with glasses that still leave a squint of 30, 40, or 50 degrees, which is unchanged or increased from year to year.

As recently as 1921 a professor of ophthalmology wrote to the dean of the graduate studies in one of our largest universities as follows:

"It is thought by many of the leading authorities on ophthalmology that treatment should be carried on for this for many years, at least, before operation is advised."

These poor, afflicted mortals are thus carried on from year to year until *all* hope of securing a useful organ is forever lost. The shame is with us ophthalmologists and it should be remedied.

The nonoperative treatment of strabismus can best be presented under the following headings:

(1) Constitutional;
(2) Optical correction;
(3) Visual training;
(4) Training of binocular vision, or the fusion faculty.

Constitutional Treatment.—It must be at once manifest that any contributing constitutional disorder should receive proper attention. I feel certain that this phase of our squint treatment has not yet received the attention that its importance deserves.

When we review the many factors which have been mentioned as cause of squint, we must admit that comitant squint is not alone a manifestation of an ocular condition, but is frequently the sequel of some frank breakdown of the general system. I think we are prone to pay too little attention to the parent who gives as the cause of squint in his child whooping cough, measles, general nervousness, etc.

It must be admitted that the general physical condition of the squinting child is usually far below par, and I can scarcely recall a case of squint in whom the general nervous system was even up to a low normal.

Mr. Inman[5] says he had thought that the child might have developed a squint to replace "emotional stress." He says:

"The profession accepted without demur the deliberate suppression of the vision; why not accept the theory of the deliberate though unconscious production of a squint?"

Mr. Inman thought there was some similarity to hysterical lesions in squint. He had observed that squinters were much more frequently afflicted with some nervous stigma, such as stammering.

Mr. Harrison Butler[6] had found squint more frequent in subjects with some kind of an emotional "kink."

Javal[7] has noticed the nervous element in squint cases, and has even gone so far as to say that it is not of any great importance to measure the degree of squint, as the degree of squint varied so much under different nervous influences which happened to be present.

When we consider squint from this broader view, it becomes evident that we are treating a patient and not an eye only. It is then our duty first of all to call in consultation the proper medical expert, and to place our patient in the best possible physical condition. Toxic conditions must be removed, whether they be in the teeth, tonsils, or intestines. The alimentary tract and the nervous system must be cared for, and where anemia, chorea, or rickets is found, appropriate remedies must be applied. In fact, a patient with a squint is an abnormal patient, and he should receive a thorough physical and neurologic examination. When this is systematically done, we will find that a very high percentage are both physically and neurologically below normal. This is our first duty to our squint cases.

Optical Correction.—Buffon in 1743 suggested the use of glasses in squint. Böhm[8] in 1845 used glasses in certain cases of squint and noticed that some saw better at a distance with convex glasses. The use of glasses as a definite relief for squint was introduced by Donders in 1864. In his "Anomalies of Refraction and Accommodation," he definitely established the beneficial effect of convex glasses in the cure of squint.

The optical correction of squint is our second step in the treatment of any case. After the constitutional examination has been made, we should next direct our attention to the error of refraction. In the chapter on examination of the patient the method of determining the refraction has been discussed, so we shall here add only a few remarks.

The child's refraction should be most carefully determined by means of a retinoscopy (skiascopy) done after he has been under atropin for at least seven to ten days. If, on account of the improper use of atropin, or misconduct on the part of the child, the surgeon cannot secure a satisfactory retinoscopy, he should repeat the examination on another day, and persist until he can secure a satisfactory one. This in very young children is our only means of securing the refraction, and its importance cannot be too strongly impressed upon the reader. The entire future of the case may depend upon securing an accurate refraction. The wrong glass at this delicate age may be the one thing that turns the tide the wrong way.

After the retinoscopy has been properly done, glasses should be ordered which will correct within 0.50 D. of the full hyperopia and all the astigmatism. This is the rule as given by C. Worth, and I have found it very satisfactory. I have found some modifications of

this rule to work well *in some cases.* Where the lenses obscure the vision markedly, it is at times advisable to order the first lenses somewhat weaker and change them for stronger ones after one or two months' use. A good plan, where the accommodation refuses to relax, is to keep up the use of the atropin and use the full correcting glasses at the same time.

Inasmuch as some of these squint cases, while using their glasses, are able to keep their eyes straight when looking at a distance, and squint only when looking at near objects, I have been in the habit of ordering about 2 D. Segment to relieve the excess accommodation, and find that they are thus enabled to use their eyes for near vision without squinting. Dr. E. Maddox[9] mentions the fact that *convergence tends to lessen on looking upward,* and has used frosted glasses in the lower part to obscure vision. He also recommends bifocals. Dr. Maddox calls attention to the fact that proximity calls forth a stimulus to the convergence reflex, and suggests that these young squinters should be prevented from looking at objects too close. This was recommended by Sichel in 1855.

It has not been my experience that glasses stronger than the error of refraction are well borne, nor that they are of any particular benefit; however, they are recommended by some.

Visual Training.—Visual training has as its object the improvement of the acuity of vision in the deviating eye. This is accomplished by means of some appliance to inhibit the use of the good eye and thus force the use of the squinting eye, or by the use of atropin in the good eye to so blur its image that the use of the deviating eye will be preferred. There have been devised many ingenious contrivances to force the use of the squinting eye, and thus bring up the acuity of the vision, or prevent the loss of vision, in the squinting eye. As early as the seventh century Paulus Aegineta devised a mask with central perforations which was to be worn as a goggle. Ambroise Paré, in 1509 (see Fig. 1 in chapter on History of Strabismus), had constructed of horn a pair of goggles with a central perforation to be used by squinters.

E. Darwin,[10] in 1778, recommended a gnomon to force the use of the squinting eye. The following, taken from the Philosophical Transactions of 1778, is of interest:

"A gnomon of thin brass was made to stand over his nose, with a half circle of the same metal to go round his temples. These were covered with black silk, and by means of a buckle behind his head, and a crosspiece over the crown of his head, this gnomon was managed so as to be worn without any inconvenience, and projected before his nose about two inches and a half. By the use of this gnomon he soon found it less inconvenient to view all oblique objects with the eye next to them, instead of with the eye opposite to them."

Darwin also recommended that a piece of gauze be worn over the good eye to obscure the vision in order to force the use of the squinting eye.

The profession is indebted to Javal[11] for the demonstration of the value of occluding the good eye in developing the vision of the squinting one. As early as 1856 Javal reports a case in which the acuity of vision was improved and the patient finally cured of her squint, the chief means of cure being the occluding of the sound eye, and preventing the use of the eyes for near.

Javal[11] has the following interesting remarks to make with reference to the occlusion of the good eye:

"In permanent convergent squint, if the squinting eye has lost the faculty of direct fixation, one can attempt to awaken it by bandaging the good eye for several weeks."

On page 90 he says:

"The principal point, on which I do not know how to insist enough, is the necessity of absolutely permanent use of the louchette. At the beginning of my researches, seeing patients every day, I noticed on Monday a state less favorable than on the Saturday preceding, and on investigation, I learned that on Sunday, the maternal vanity getting the upper hand, the two eyes were left uncovered for several hours, to attend mass, or make a visit."

E. Landolt also advocated bandaging the good eye in order to force the use of the squinting one. To C. Worth[12] much credit is due for his valuable work along this line.

The use of some form of occlusion of the straight eye is indicated in all cases of permanent squint except the alternating type. The indications to be met are:

(1) To reestablish central fixation.
(2) To improve the acuity of vision.
(3) To change the squinting eye to the fixing eye.
(4) Restoration of binocular vision.

The most rapid and effective way of reestablishing central fixation, where this is lost, is by absolute occlusion of the good eye to thus force the use of the weaker one. This is best done by placing over the good eye a very snug eye pad with bandage. This should be kept up constantly from four to six weeks, and the acuity of vision as well as the ability to fixate observed from time to time. It has been my observation that, where this rule is followed, within four to six weeks a very definite fixation is developed and also a very definite improvement in the visual acuity has taken place. This is diagnostic. If no improvement of vision in the amblyopic eye is noticeable, and the power of central fixation has not been secured, it is of little benefit

to continue longer, and in these cases one can expect only a cosmetic result.

As soon as the visual acuity has been brought up to nearly normal or even up to 6/12, the use of atropin in selected cases can now be substituted for the occluding bandage. I feel that we are justified in the use of the occluding bandages for two reasons: First, it will develop central fixation and better vision in a large percentage of the cases. Second, it is diagnostic, in that it points out these cases where amblyopia is congenital or pathologic, and not due to functional depression; i.e., amblyopia ex anopsia.

Use of Atropin.—We are again indebted to Javal[13] for this valuable remedy in the treatment of squint. He says:

"One has seen that atropin, instilled for a sufficiently long time and in strong doses in the better eye, often makes the variable part of squint disappear (obs. 18, p. 19), and when associated with the use of convex glasses, a cure of these cases where there is no fixed deviation."

On page 75 he says:

"In periodic hypermetropic convergent squint, it happens that the better eye, having trouble because of atropinization, forsakes fixing and accommodating; then the other eye rights itself and the squint disappears as if by enchantment."

Atropin is indicated in all cases of squint to paralyze the accommodation to determine the error of refraction. In the treatment of squint it is indicated:

(1) In both eyes to break the spasm of accommodation and to relieve the excessive convergence.

(2) To paralyze the accommodation in the fixing eye and force the use of the previously squinting one.

It is sometimes necessary to use atropin for more than ten days to secure a complete relaxation of the accommodation and undo the bad habit of excessive convergence formed by the squinters. In many cases of recent squint it is well to use atropin in both eyes after the glasses are ordered. This sometimes aids materially in breaking the squint habit, and it teaches the child to depend upon his glasses. Worth has warned against the too prolonged use of atropin and considers it one of the best ways of producing a permanent squint, as it is his opinion that it tends to cause a more complete suppression of the image of the already squinting eye.

In order to cause the child to use the crossed eye, a drop of one-half per cent solution is used in the better or straight eye night and morning.

If atropin causes the vision in the straight eye to be less acute than that in the deviating eye, the latter will at once straighten and will be the eye used. This use of the deviating eye will usually bring about the desired acuteness of vision. If we could secure 6/6 or normal vision in the deviating eye, there would usually be very little trouble in securing binocular vision, and thus we would be able to cure our case without operating. In some cases in which the deviation occurred very early, or in which the deviation has persisted for several years, the use of atropin does not sufficiently interfere with the vision in the good eye to make that vision as poor as the vision in the deviating eye. In this class of cases it is necessary to pad or cover completely the good eye and to force the use of the deviating eye in order to develop the macula in that eye. Hence our first step in the use of atropin to force the use of the deviating eye; where that fails, we must bandage the good eye and exclude it entirely from use until the vision in the deviating eye has improved to such an extent that it will be used when the good eye is atropinized. Occluding the good eye might be practiced for a certain number of hours each day; but in cases in which amblyopia is marked it may become necessary to occlude the good eye for weeks at a time, as indicated above. If the vision of the deviating eye has been brought to 6/12 or better, a full correction of the error of refraction will usually cause the eyes to remain straight in cases of slight degrees of deviation.

In patients with slight refractive defects, the use of atropin does not usually blur the vision sufficiently to cause the use of the squinting eye, hence the occlusion treatment will be necessary. Atropin in the good eye is only effective in those cases in which it causes the use of the squinting eye.

In cases where the previously fixing eye becomes the squinting eye, it will become necessary to stop the use of the atropin after a time, and may even become necessary to use atropin in the previously squinting eye in order to cause the other one to be used. This class of cases can always be cured, and a high percentage of these cases can be cured without operating.

Training of the Binocular Vision or Fusion.—No set of eyes can be put straight and kept straight without binocular vision or fusion. This faculty, innate in man, is the *one essential* in curing squint; hence the importance of determining its presence, its degree, and adopting methods to increase its amplitude in each case. Worth has given us three grades of binocular vision, as follows:

First. *Simultaneous macular perception;* that is, the ability to recognize objects perceived by each macula. In these cases there is no particular desire for binocular vision. In other words, the fusion

power is of the lowest grade, and any contributing cause may upset the binocular vision and cause squint.

Second grade comprises those who have *fusion* with some amplitude. In these cases there is a definite desire for binocular vision, and when similar objects are placed in the stereoscope or the amblyoscope these objects are readily fused into one image when approximated, and this one image is maintained even when the stereoscope or amblyoscope is separated several degrees. This shows that there is a definite effort on the part of the patient to fuse these images. In these cases the phoria is overcome in the interest of binocular vision.

The third grade Worth calls the "sense of perspective." Each eye sees an object at a different viewpoint from the other eye. In other words, an object seen by the right eye is seen farther around to the right than the same object is seen by the left eye, and vice versa, but these images are so blended that we are cognizant of but one mental impression. This sense of perspective enables us to determine depth and fullness of objects and cannot be obtained with the use of one eye only. If we now add perfect acuity of vision, we shall have such a strong and perfect binocular vision that no amount of refractive defect or overacting muscles can cause squint. In fact, as Worth has aptly said, "Nothing but a paralysis of the ocular muscles could cause squint, and this would produce an intolerable diplopia."

Test for Binocular Vision.—One of the best tests for binocular vision is the Snellen's colored-glass test, or better known as the "four dots test," as described by Worth.

This consists of two green lights, one red light, and one white light placed in a frame which is placed in a window, or in front of some bright light (Fig. 53). As a pure red glass will allow only red rays of light to pass through it, and a green glass will permit only green rays, the light which is passed through the red glass cannot be seen through the green glass, and vice versa. In making the test with these different colored glasses, the patient is seated about twenty feet away and a trial frame with the red glass before the right eye and a green glass before the left is adjusted to his face and he is directed to look at the lights. We now ask him what lights he sees. If he says he sees two dots, he is using the right eye only. If he sees three dots, two green and one white, he is using the left eye only. If he sees four dots, two green, one white, and one red, he has at least grade one of binocular vision. If he sees five dots, two green, one red, and two white, he has diplopia. This is also a very good test for malingering.

One of the most convenient and at the same time one of the most exact methods of testing binocular vision is the use of Worth's amblyoscope (Fig. 54).

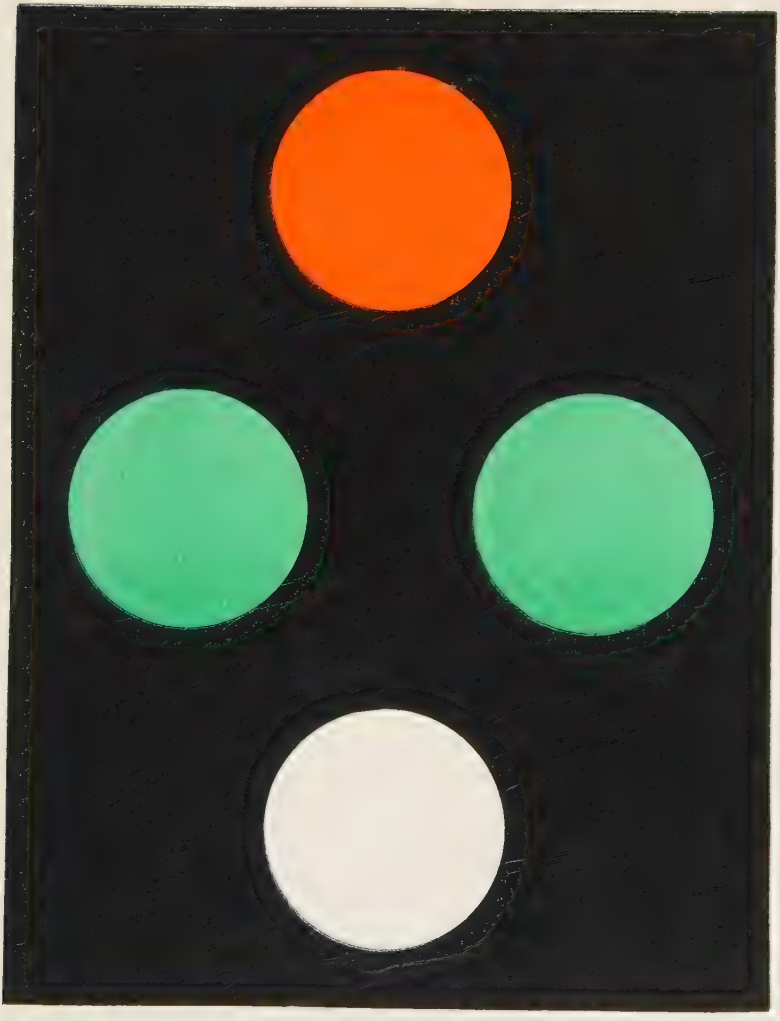

Fig. 53.—Four dots fusion test. (From Worth, *Squint.*)

This instrument is to be adjusted for orthophoria, and various sets of diagrams as recommended by Worth are placed in the instrument, and the child is asked what he sees. For instance, if a star is placed in one end of the amblyoscope and a half moon in the other, we can easily determine by his answers whether he is using both eyes. (See Figs. 63 and 64.) If he can see both the star and the moon, he has first grade binocular vision. If pictures, as Figs. 65 and 66, be placed in the amblyoscope, one can readily determine whether he has second grade of fusion, according to whether or not he sees the complete picture.

Fig. 54.—Worth's amblyoscope.

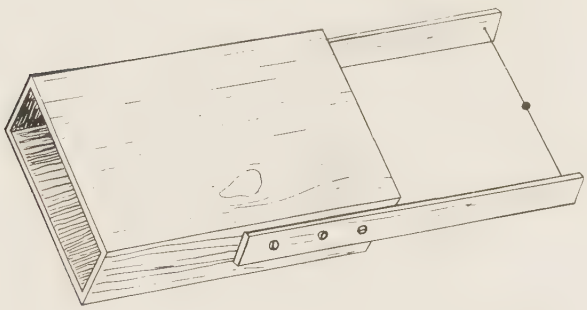

Fig. 55.—Hering's drop test. (From Worth, *Squint.*)

If Fig. 67 is now placed in the amblyscope, and the patient is able to recognize the sensation of depth as though he were looking into a large cylinder or barrel, he has the third grade of binocular vision, or the *sense of perspective.*

Hering's drop test for the sense of perspective is one of the oldest and one of the best (Fig. 55). Its use, however, is time-consuming, and as we can secure the information which it gives with more simple means, its use is now largely abandoned.

Bar-reading Test.—Javal introduced the bar-reading test, which consists of placing one or more metal bars about one centimeter in width half-way between the patient's eyes and the page, and requir-

ing him to read. If the patient can read without *hesitating or moving the head,* he is using both eyes. (Fig. 56.)

A very simple test for determining binocular vision is the use of a lead pencil. A pencil or penholder is held between the patient's eyes and a reading test, and the patient is instructed to read. If he can

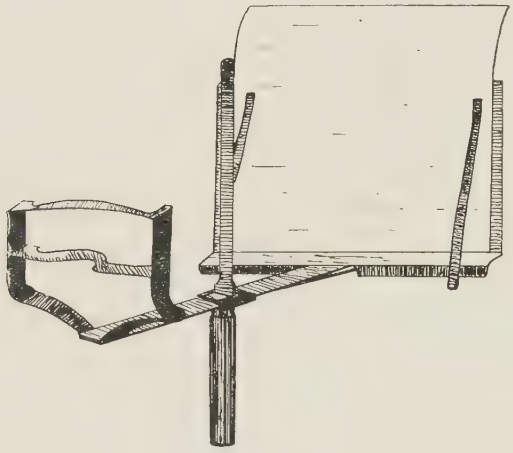

Fig. 56.—Javal's bar reading test. (From E. Javal, *Manuel du Strabisme.*)

Fig. 57.—Triple control bar reading device. (From Wells, *The Stereoscope in Ophthalmology.*)

do so without any movement of the head, binocular vision is present. (Fig. 57.)

A knowledge of the presence or absence of binocular vision is necessary in each case. It gives us a fairly definite idea as to the line of treatment that will be necessary to follow and also enables us to prog-

nosticate with more accuracy what the final result will be. If a recent case of squint has fairly good visual acuity with at least grade two of binocular vision, persistent training with the amblyoscope, and constant use of the proper glasses usually give satisfactory results. If no grade of binocular vision is elicited, and a marked degree of squint is present, it becomes a very different matter and will probably call for early operative interference, and the end-result may not be more than cosmetic.

Method of Training Binocular Vision.—Failing to obtain binocular vision by means of the use of atropin, glasses, and padding the eye, we now resort to the use of some form of the stereoscope for orthoptic treatment.

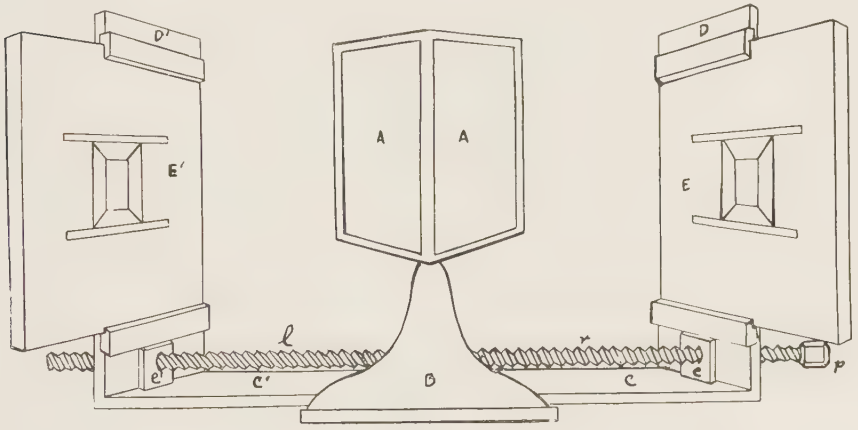

Fig. 58.—The Wheatstone stereoscope. (*The American Encyclopedia of Ophthalmology.*)

The Stereoscope.—Since some form of the stereoscope is our chief agent in the training of binocular vision, it will be of interest to follow its development.

Wheatstone, in 1838, before the British Philosophical Association, presented a paper on the Physiology of Vision, in which he described the first stereoscope, and gave to it the name of "stereoscope" (stereon, *solid*, and scopein, to *see*). The chief function of the stereoscope is the blending of the two images of an object taken from slightly different angles, and thus giving to the objects the appearance of solidity, or a sense of *relief*. Wheatstone's stereoscope was large and clumsy, but was of great value in introducing the idea of stereoscopic vision. (Fig. 58.)

In 1849 Sir David Brewster invented a lenticular stereoscope (see Fig. 59) which was a very practical instrument, and from which many modifications were afterwards made.

In 1852 Wheatstone[14] presented a second paper on physiology of vision, in which he had the following to say:

"At the date of the publications of my experiments on binocular vision, the brilliant photographic discoveries of Talbot, Niepce, and Daguerre had not been announced to the world. To illustrate the phenomena of the stereoscope I could, therefore, at that time, only employ drawings made by the hands of an artist. Mere outline fig-

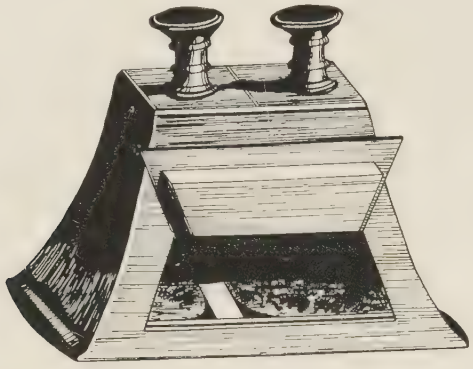

Fig. 59.—Brewster's lenticular stereoscope. (*The American Encyclopedia of Ophthalmology.*)

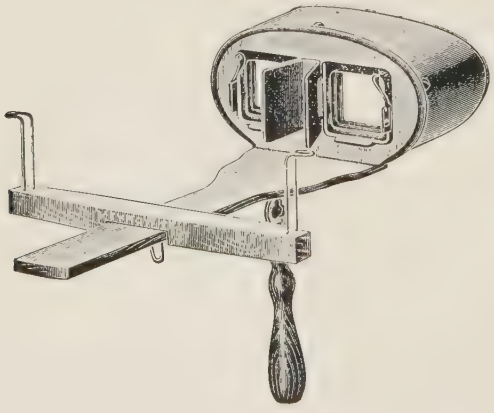

Fig. 60.—Holmes' stereoscope with clips for extra prisms. (From Wells, *The Stereoscope in Ophthalmology.*)

ures, or even shaded perspective drawings of simple objects, do not present much difficulty; but it is evidently impossible for the most accurate and accomplished artist to delineate by the sole aid of his eye the two subjects necessary to form the stereoscopic relief of objects as they exist in nature, with their delicate differences of outline, light and shade. What the hand of the artist was unable to accomplish the chemical action of light, directed by the camera, has enabled us to effect. It was at the beginning of 1839, about six months after the appearance of my memoir in the Philosophical Transactions,

that the photographic art became known, and soon after, at my request, Mr. Talbot, the inventor, and Mr. Collen (one of the first cultivators of the art) obligingly prepared for me stereoscopic Talbotypes of full-sized statues, buildings, and even portraits of living persons."

In 1856 Wheatstone published a book in which he outlined the principle of the stereoscope, and gave complete data as to stereoscopic photography.

Holmes' Modification of Brewster's Stereoscope.—Dr. Oliver Wendell Holmes modified the Brewster stereoscope, and introduced it to the American public. He coined the word, "stereograph," for the double picture designed for the stereoscope (Fig. 60). Dr. Holmes did not use the stereoscope for the treatment of squint, but expected it to be used for pleasure and educational purposes. Concerning it, he wrote as follows in the Atlantic Monthly:[15]

"We write principally to wake up an interest in the new and inexhaustible source of pleasure, and only regret that the many pages we have filled can do no more than hint the indefinite resources which the new art has laid open to us all."

Holmes' prophecy with regard to its aid to science has been abundantly fulfilled, as some modification of the stereoscope is now used in many branches of science.

LeConte, in 1881, published a book on *Sight,* in which he explained the theory of binocular vision. He quotes Brucke as follows:[16]

" 'In regarding a solid object or two stereoscopic pictures in a stereoscope, the eyes are in incessant unconscious motion, and the observer, by alternately greater and less convergence of the axes, combines successively the different parts of the two pictures as seen by the two eyes, and thus by running the point of sight back and forth reaches by trial a distinct perception of binocular perspective or binocular relief, or depth of space between foreground and background.' "

It was LeConte's opinion that the "mind perceives relief instantly, but not immediately, for it does so by double images." Parinaud (Annals d'Oculistique) does not agree with Brewster and Brucke, who maintained that stereoscopic fusion and binocular vision were identical. Of this he says:[17]

"Stereoscopic vision is obtained not by the fusion of the figures themselves, but by the fusion of the virtual images of the figures."

The first definite use of the stereoscope in the treatment of squint which we have been able to find was made by Javal about 1863. In his Manuel du Strabisme, page 1, he says:

"In 1863, after Giraud-Teulon had tried without success to cure the squint with prisms, I had the idea of using the stereoscope. It was

then I met with *repulsion of images* * * * and it was then also I took account of *neutralization,* thanks to which squinters do not see double, and I noticed the usefulness of the stereoscope to awaken diplopia."

Javal was certainly first to introduce the stereoscope as a very definite aid in the cure of squint. He devised two stereoscopes for the treatment of strabismus. His first was the better. It had five movements, and was a very efficient instrument for low degrees of defect. His hinged stereoscope was adopted for stereoscopic training of higher degrees of squint. This instrument was also used for measuring the angle of squint.

Javal recommended the use of the stereoscope to train the fusion, and says (p. 31, l.c.):

"We are being led to attribute a large part to the education of the faculty of fusion of the images received by the two eyes."

He was one of the first to arrange a set of cards of different grades for the purpose of training the fusion. Many of these are still in use. He recommended the use of the stereoscope especially after operation.* (See Javal.[25])

His method was to correct the error of refraction with glasses, use atropin in the good eye, exclude entirely for a time the good eye in selected cases to develop the vision in the deviating eye, use the stereoscope to bring about fusion, where this is possible without operating, and in those cases in whom the deviation was too great, he recommended operation, early operation, to set the eyes sufficiently straight to use the stereoscope. With reference to operating, he says (l.c., p. 158):

"It is a mistake to believe that operation gives better results at a more advanced age. The sooner it is done, the greater are the chances that it will suffice."

The present-day treatment offers but little in advance of his methods of thirty years ago. He says (p. 150, l.c.):

"When the squint with which one has to do is moderate, that is to say, when it is possible to obtain fusion in the stereoscope, it is then that I begin treatment, believing that the attraction of the double images thus perfected will be a great aid in the perfect cure, immediately after the operation. But, for a few where the deviation is so as to make operation intervention inevitable, I prefer not to wait, for the operation launches diplopia and permits the exercise of stereoscopics by means of the ordinary stereoscope, rather more crude than the stereoscope of five movements."

Landolt used a very simple stereoscope (Fig. 61) which he devised

*He usually did a tenotomy.

about 1870 and described in his masterpiece, *The Refraction and Accommodation of the Eye*, 1886.[18]

Landolt[19] was still using this stereoscope with success up to a few years ago. Of it he says:

"We employ for the reestablishment of binocular vision a very simple apparatus. It consists of an ordinary stereoscope box, from which we have removed the prisms, in order to substitute for them whatever glasses from the trial case may be desirable, whether spheric, cylindric, prismatic, or a combination of them.

"It is well to make the partition of the stereoscope rather long in order to prevent one eye from trespassing on the visual field of the other. The visual objects placed at the opposite end of the box from

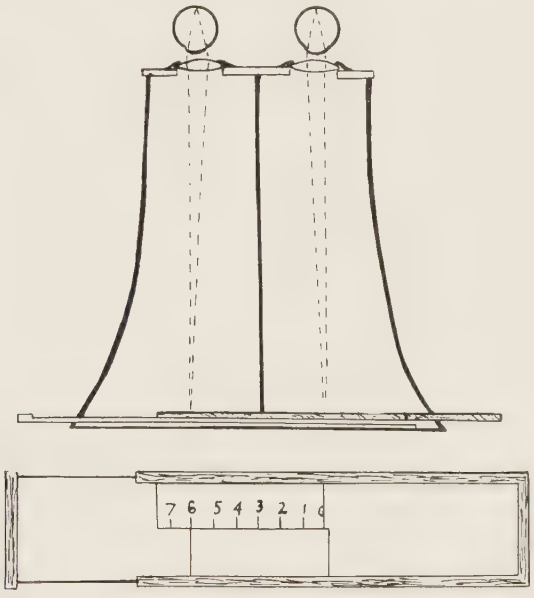

Fig. 61.—Landolt's stereoscope. The upper part of the figure represents the stereoscope box furnished with glasses which adapt each of the two eyes to the distance of the visual objects.

These latter are fixed to two boards which slide one upon the other in such a way that one can alter at will the distance between the two figures. This distance is indicated in millimeters by the division of one of the boards.

The figures to be fused are represented here by two simple red lines, the one corresponding to the point zero, the other to the sixth centimeter of the division. (*American Encyclopedia of Ophthalmology.*)

the eyes to be exercised may be brought nearer to or separated from each other at will. They may even be raised one above the other, or inclined relatively to each other.

"The principle of any stereoscope useful for the treatment of strabismus is that the instrument employed places the eyes in the conditions which are most favorable to the fusion of their retinal images.

"One of the eyes being generally weaker, or in any case less exercised, than the other, it is a question, first of all, of making its visual impressions as vivid as possible. For that purpose we correct its astigmatism and adapt it with perfect precision to the distance of the

object which it is to see, in order to procure for it an absolutely clear retinal image. After doing so, we must seek the relative position of the two figures in which their fusion is possible * * *.

"But the greater part of the time the patients do not fuse, whatever may be the interval between the two objects. They see alternately the one or the other, when the vision of the one eye is about as good as that of the other, or only the object corresponding to the better eye when the vision of the two eyes is different.

"In this case it becomes a question of attracting the attention of the weaker eye to the object which corresponds to it, to cause it to fix energetically, excluding its congener. By successively covering and uncovering the better eye for longer or shorter intervals, indeed, even by furnishing it with a too strong convex glass which lessens the clearness of its vision, one may succeed in causing the patient to see the two objects simultaneously, and at length to fuse them into a single one. Much patience is required, in order to achieve this result, on the part of the surgeon as well as of the patient. It is for this reason that this training, so logical and useful, is far from having found in ophthalmic practice the place which it deserves."

Landolt and also Priestly Smith devised a set of fusion tubes the object of which was to train the fusion and overcome the tendency to suppression of the image of the deviating eye. By means of these tubes, the image of the weaker eye could be made brighter, and vice versa.

Bishop Harman, of England, has introduced an instrument which he calls the *Diaphragm Test,* which is a reversal of the Javal bar-reading test.

Probably one of the best instruments for fusion training, and the one most used in this country, is the Worth amblyoscope. Claude Worth, of London, 1895, perfected a stereoscope which he called the amblyoscope—"An instrument by means of which a nonseeing eye is trained to take its share in vision." (Worth.) The following description of the instrument is taken from C. Worth's book on squint, page 122:[20]

"The instrument consists of two halves joined together by a hinge. Each half consists of a very short brass tube joined to a longer tube at an angle of 120°. At the angle of junction of the tubes is an oval mirror, protected on the outside by an oval plate of brass. Each half of the instrument has at its distal end an object-slide carrier, and at its proximal end a convex lens having a focal length of five inches—the distance of the reflected image of the object-slide. In front of each lens is a slot into which a prism, axis vertical, may be inserted if required. The diameter of the tubes is 1½ inches.

"A brass arc connects the two parts of the instrument, being clamped on one side by a binding screw set in a long slot and on the other by a binding screw set in a short slot. When the screw in the long slot is loosened, the two parts of the instrument can be brought together to suit a convergence of the visual axes up to 60°, or separated to suit a divergence of as much as 20°. When this screw is

tightened and the screw in the short slot loosened, an amplitude of movement of about 10° only is permitted."

Nelson Black, of Milwaukee, has added a vertical adjustment to Worth's amblyoscope, which is of great value when hyperphoria is present.

Worth had constructed a light carrier with two movable lights, by which one is able to adjust the degree of luminosity needed to bring out the image of the defective eye or to decrease the brightness of the image of the better eye, so as to make them appear as nearly the same for the purpose of fusion. These lights are adjusted in front of the two ends of his amblyoscope when in use. A couple of bracket lights can be used very conveniently for the same purpose. One can accomplish much with the use of one lamp or by using the light from

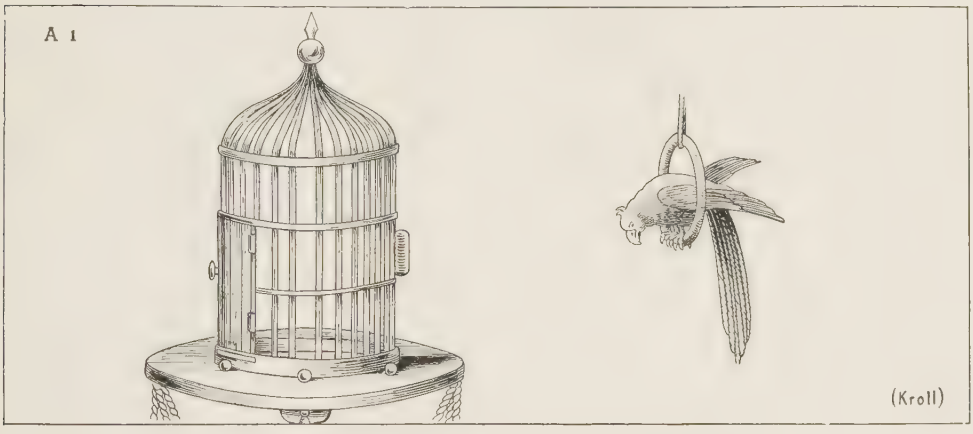

Fig. 62.—Test for first grade fusion. (From Worth, *Squint.*)

a bright window, so placing the patient as to give advantage to the weaker eye.

Worth has devised a very attractive set of object slides to be used in his instrument. These are designed to be used according to the degree of fusion present.

I have found those shown in Figs. 62-67 very serviceable.

Worth divides his object slides into three classes:

1. Those which do not require any blending of images, i.e., those in which there is only simultaneous vision—his first grade of fusion, such as Fig. 62.

2. Those requiring true fusion of images in order that a complete picture can be seen. This is his second grade of fusion. These are represented in Figs. 63, 64, 65, and 66. It will be observed that the pictures are similar except for certain omissions in each slide, so that it requires both images to make a complete picture.

3. Those which are intended to bring out the third grade of fusion, or a sense of perspective, as Fig. 67.

The following lucid description of Worth's method of fusion training is so well presented that we shall quote it at some length:[21]

"Seated in a chair facing the lighting apparatus, one takes the child on one's knee and adapts the amblyoscope roughly to the angle of his squint. One puts in the instrument first a pair of object-slides, which require no fusion, only simultaneous perception—those shown in Fig. 62, for example. Each light is at first about four feet away from its object-slide. Suppose the cage is before the fixing eye and the bird before the squinting eye. The child, on looking into the

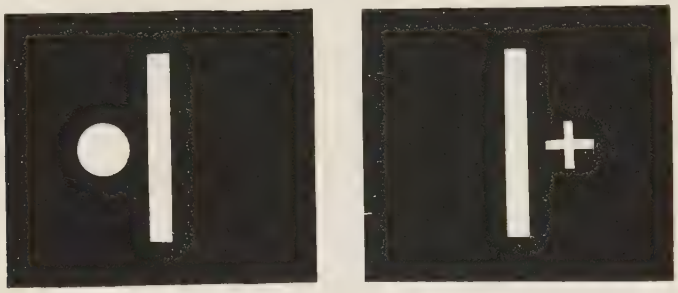

Fig. 63.—Slides for fusion exercise. (From Worth, *Squint.*)

Fig. 64.—Slides for fusion exercise. (From Worth, *Squint.*)

amblyoscope will see only the cage. He is told to look for the bird, while the light before the deviating eye is brought nearer and nearer. At last a point is reached when the illumination of the object-slide before the deviating eye becomes so intense that the vision of this eye can no longer be suppressed. The child suddenly says he sees the bird he was told to look for. But he has now usually lost sight of the cage before the fixing eye. The relative distances of the lights are then readjusted until, after a few minutes of alternation of the vision of the two eyes, the child sees the bird and the cage simultaneously. The child is then allowed to grasp the amblyoscope with both hands, while the surgeon, putting his hands over the child's, converges and diverges the two halves of the instrument, in order to make the bird appear to go in and out of the cage. Other similar pairs of object-slides are then shown. The child soon learns to move the instrument

himself, so as to put the bird in the cage, the cat in the chair, the clown in the hoop, etc. One must talk to the child all this time, as it is only by encouraging him to chatter that one learns what he really sees.

"A pair of slides requiring fusion of images is now shown—Fig. 65, for example. The child at first sees two men, each picture being imperfect. Soon a position is found in which the child sees one man

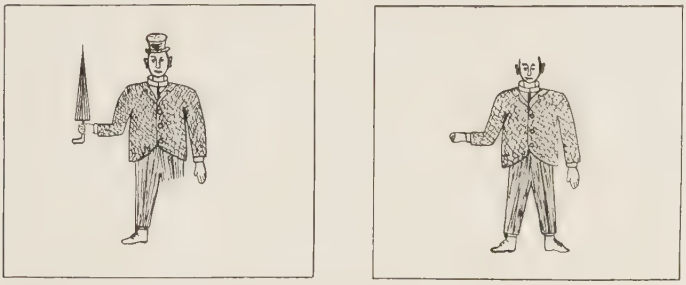

Fig. 65.—Slides for fusion exercise. (From Worth, *Squint.*)

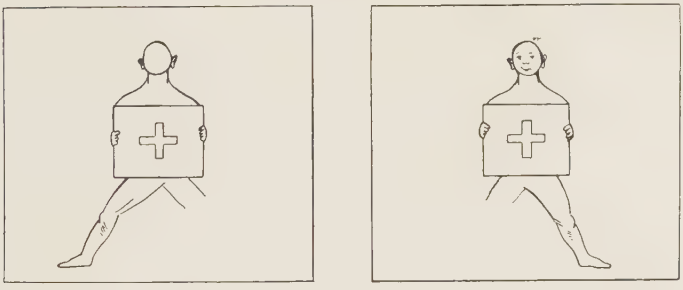

Fig. 66.—Slides for fusion exercise. (From Worth, *Squint.*)

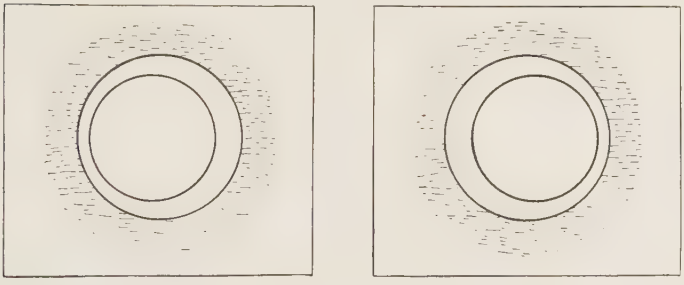

Fig. 67.—Test for third grade fusion. (From Worth, *Squint.*)

having two legs and a hat. The binding screw in the long slot is now tightened and that in the short slot loosened, so that the amplitude of movement between the two halves of the amblyoscope is restricted to about 10°. Other pairs of slides requiring fusion of images are successively shown the child, and he is encouraged by one's remarks to examine every part of the fused picture. After a time it is found that the angle of convergence of the instrument may be varied slightly

without the fused picture coming to pieces. The child has now, under these special conditions of illumination and convergence, the second grade of binocular vision—true fusion with some amplitude.

"The next step is to increase the amplitude of fusion. The intensities of the lights and the angle of convergence of the amblyoscope are arranged as before. Devices requiring fusion, such as Figs 65 and 66 are shown. An attempt is made gradually to diverge and converge the two halves of the instrument, more and more, while the child is examining and talking about the various pictures shown him. After some practice, in the case of a young child, a considerable range of movement becomes possible, fusion being still maintained. This

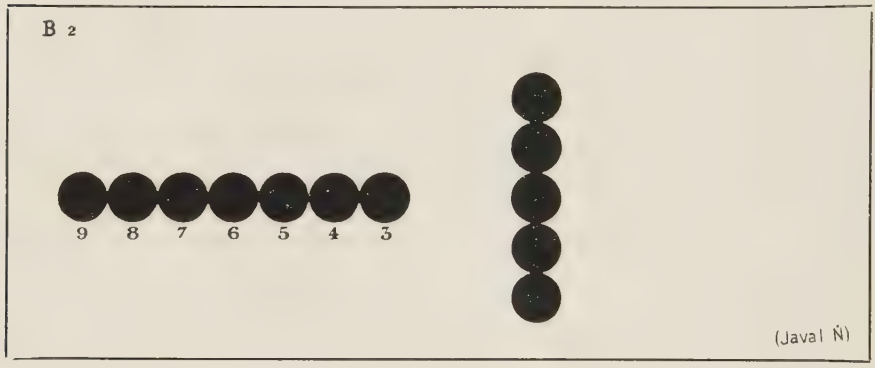

Fig. 68.—Stereographs for fusion convergence. (Javal.)

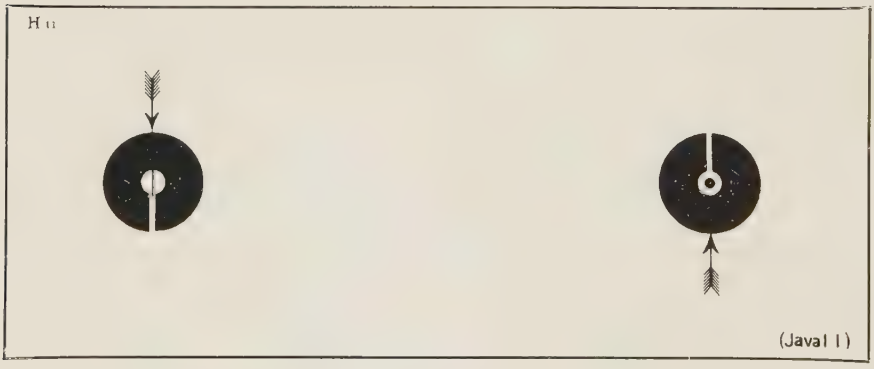

Fig. 69.—Stereographs for fusion training. (From Javal.)

'amplitude of fusion' may, for practical purposes, be taken as a measure of the extent to which the fusion faculty has been developed.

"A child who has any considerable amplitude of fusion will nearly always be found to have acquired the third grade of binocular vision also—the sense of perspective. The slides, Fig. 67, are shown, and the child is asked whether he is looking at the outside or the inside of the tub. He will at once say 'the inside.' If these slides are now changed from one tube to another, he will see the tub bottom up.

"The child having now acquired the highest grade of binocular vision under these special conditions of illumination and convergence, the next step is gradually to equalize the light before the two eyes.

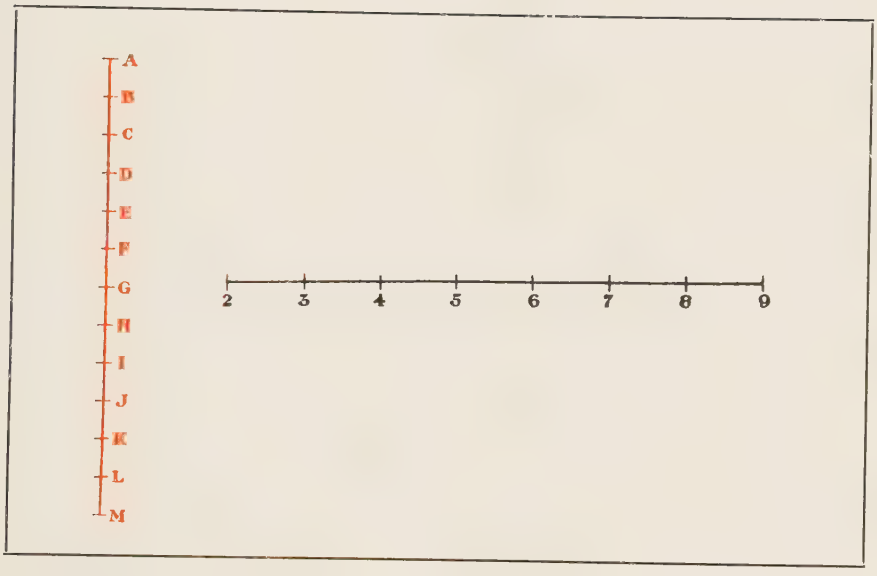

Fig. 70.—To measure stereoscopic hyperphoria. (From Wells.)

Fig. 71.—For fusion and convergence training. (From Wells, *The Stereoscope in Ophthalmology.*)

This may readily be done, at this stage, without a return of the suppression.

"If the child be young enough, a fairly strong 'desire for fusion' may usually be created in five or six lessons, given at intervals of one week. If the training can be efficiently carried out at home, the child should have a lesson of a few minutes' duration every morning."

Worth stresses the importance of beginning the fusion training "at the earliest possible age. I repeat this because it is of extreme importance—the only key to success." He claims to be able to begin training the fusion as early as three years of age. In selected cases this is possible, but in many intractable children this becomes a difficult procedure, if not an impossible one. I do feel, however, that he is eminently correct in claiming that fusion training should be begun "at the earliest possible age," and I also agree with his claim that little real progress in fusion training can be made after seven or eight years of age. Of this, he says (p. 121, l.c.):

"After the age of six, or at the latest, seven years, the results are seldom worth the time and trouble they cost."

Javal was much more enthusiastic about getting results in older subjects, but he also admitted it required much time and patience, even requiring treatment daily for years. In this age of acute haste, neither the physician, the patient, nor the parent could ever endure such a siege.

One of the best methods of fusion training is that devised and recommended by Dr. David Wells, of Boston. In 1904 he published a selection of stereoscopic cards which were taken largely from those devised by Knoll, Hale, Javal, and Dohlfield. To these he added some new ones. These cards are still in use, and are one of the best selections published. In 1902 Wells had a stereoscopic attachment with adjustable carrier added to his phoro-optometer. This instrument is one of the best fusion-training stereoscopes on the market. The following are some of the charts which are very useful: Wells' ON NE, with the letters N in red, is a very useful card. The dots and arrow chart is of value in measuring the fusion phoria. (Figs. 68, 69, 70 and 71.)

His stereoscope has two advantages over most stereoscopes. First, it permits prismatic exercises by means of decentering strong lenses, and secondly, it permits the operator to watch both eyes while the patient is taking his exercises, thus enabling the surgeon to detect alternate vision or suppression.

The use of decentering of lenses for fusion training is especially emphasized by Wells, as it permits a more gradual separation of the images, or a tendency to a separation, and enables the patient to hold the images together longer.

Wells' description of the decentering effect is as follows:[22]

"With o.u. plus 10., cards at 10 cm. are in focus. If the separation corresponds to that of the pupillary distance of the patient's eyes, no prismatic element is exhibited. If decentered 1 mm., 1Δ approximately is produced. Thus, plus 10. spheres make the calculation of the prism extremely simple. If the spheres are decentered out 5 mm., we have put before the patient 5Δ of prism base out, just as truly as though a 5Δ prism were inserted in the clip. If the spheres are decentered *in* 5 mm., 5Δ base in is obtained. As the pupillary adjustment may be varied from 50 to 75 mm., it follows that 10Δ or more may be utilized by this simple principle of decentration."

Fig. 72.—Well's Stereo-optometer.

Wells' instrument (Fig. 72) has one advantage over the Worth amblyoscope in that the eyes of the patient can be at all times under observation. Of this Wells says:[23]

"With the latter (the amblyoscope) the eyes cannot be watched, and we must depend on the patient's statements, which are naturally very unreliable, especially when treating children."

Wells maintains that the limit of fusion should be at least 80Δ, and claims to be able to train some cases to overcome as much as 100Δ,

especially where the stereoscope is used. He recommends the Holmes stereoscope, with clips for inserting extra prisms 5Δ, 10Δ, and 15Δ for use at home, and gives about twelve lessons or treatments in his office of about fifteen to twenty-five minutes, requiring that *no* interruption of any kind occur during the treatment.

Wells[24] describes Cross' modification as follows:

"Cross, of Worcester, Mass., has devised a cylinder with 14 facets, on which he has pasted 13 cards of Series H and I. This is mounted on the Holmes stereoscope, arranged with a ratchet, so that the patient can turn up one after the other of the cards, progressing in either direction."

Cross suggests that the patient's eye be closed while the cylinder is being revolved for purposes of rest. Wells has suggested that a window be cut in the hood of the Holmes stereoscope for the purpose of watching the patient's eyes, a very good suggestion. In the latest models of the Holmes stereoscope, clips are placed to receive prisms of various degrees for purposes of exercising the fusion faculty. I have found the use of these of much service in increasing the amplitude of fusion.

Wells warns against delaying operative procedure, and recommends advancement in young children in whom no decrease of the degree of squint is shown within a few months, and in those cases in which the deviation is so great as to preclude the possibility of orthoptic training being a success. He says (p. 12209, l.c.):

"A rudiment of fusion can often be trained into a refined faculty if the eyes are put approximately straight by operation."

The use of the stereoscope is particularly indicated after operative procedures. Javal[25] says:

"Two days after the operation, exercises of fusion can be undertaken, and it is here that the stereoscope renders the greatest service."

Landolt[26] says:

"Immediately after the definite removal of the dressing, exercises for binocular vision must be begun. The patient operated upon for convergent strabismus will wear spectacles to correct the total hypermetropia and astigmatism, if it be present. The one who has been operated upon for divergent strabismus will be allowed, and even urged, to look at objects at close quarters, for the purpose of exercising his convergence. All patients should go through exercises intended, firstly, to perceive simultaneously the visual impressions of both eyes, then to combine the true impressions so as to give rise to the sensation of the third dimension."

It has been my practice for years to operate early and train the fusion afterwards. (See chapters on Operative Treatment and Illustrative Cases.) By means of more accurate operative procedures, the eyes

can be set so nearly straight that the fusion training becomes much easier, and the results much more satisfactory. Cases of *true* antipathy to fusion are so seldom found that one comes to believe there are no such persons. In the few cases in which I had suspected this condition, I was able to develop enough fusion to take them out of this class. This was done by much patience and effort on my part and cooperation of the patients. It must be granted that if there were no crossing of any of the nerve fibers at the chiasm, there could never be developed any degree of fusion.

Effect of Stereoscopic Training.—It is difficult to tell just what this training accomplishes, just where the effect is produced. Wells[27] says:

"The rapid development of adduction, which is so often obtained by this so-called 'gymnastics,' strongly suggests that the gain is not a muscle hypertrophy, but an increase in innervation, either in the responsiveness of the end organ in the muscle, or the convergence center, or both. In the educational treatment of tabes the incoordination is overcome by teaching the patient to gauge his motor impulses by the eye, in lieu of the normal sensory control. Repeated artificial contractions of the internal rectus (the ciliary remaining relaxed) establish a habit of increased action, so that it no longer lags when the impulse to converge and accommodate is felt. The coordinating center may also be taught to better appreciate the advantages of binocular perspective. This is no special pleading, but analogous to other sensations. The pianist makes his fingers educate his brain, that the brain may do better work with the fingers. Tasks consciously performed are in time relegated to subconscious control. If this interpretation of muscle gymnastics be accepted, it is evident that the first indication is to teach the patient the fascination of true binocular fusion. Just as in the ordinary prism exercises, with the eye fixed on a distant point, the aversion to diplopia is an incentive to increased muscle action, so here the fused image becomes an anchor. With the eyes fastened on a fused image, made up of half pictures, one strongly resists an impulse which tends to pull it to pieces. The decentering of plus 10. lenses is a subtle means of insinuating such an influence."

We no doubt train the child's attention. We increase the innervation for binocular vision, and also increase its amplitude, just as the amplitude of convergence can be increased by exercise with *prisms*. We break up a vicious habit of neutralization, as Javal puts it. He says:[28]

"It is equally by education that it is necessary to undo the occurrence of a very curious phenomenon, to which I have given the name, *neutralization*. It is an inhibitory act which permits us to ignore certain impressions noxious to vision and particularly unfavorable to binocular vision."

No doubt the stereoscopic exercises do as much toward educating the mind away from suppression, or what some have termed *supre-*

sopsia, as it does toward educating the fusion faculty itself. It does both, however. It rids the mind of the vicious habit of suppressing the image of the previously deviated eye, excites the mind to attention to the very act of fusion itself, and, as Wells aptly puts it, "teaches the patient the fascination of true binocular fusion," besides increasing his power of fusion, both as to degree and amplitude. This general education and innervation exercise should be methodically and persistently given by the physician himself at first, to see that *no* lax or careless method is being used by the patient, and then it should be relegated to a *specially trained* assistant or nurse, whose duty it shall be to give these treatments with as much exactness and intensity as a music teacher would demand of his pupils. These should be given at first about ten minutes at a time, daily, if deemed necessary, then fifteen to twenty minutes at a time on alternate days; after sufficient progress has been made, twice a week visits with exercises at home. Worth and Jackson claim that this work cannot be done by a nurse. With the *proper* education and the early supervision of her work, with a check-up from time to time, I feel the *very best* work can thus be secured. Make it the nurse's real job, and she will become really interested in it, and will strive to secure definite results. The very busy oculist will seldom give this pedagogic treatment, and the nurse by nature and education is better fitted to do it.

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CHAPTER XI

OPERATIVE TREATMENT OF STRABISMUS

At the very outset, let us ask ourselves what is the object to be attained when one undertakes to correct strabismus by means of a muscular operation. E. Landolt[1] has ably and lucidly answered this question for us as follows:

"Now, in regard to these muscles, we should, in the first place, never forget that all the muscles of the eye form a whole, which is in close relationship with a corresponding whole in the other eye. Together they form the *binocular motor system.* To insure binocular vision, this system causes both eyes to move in harmony as if they were one single organ, 'the double eye' of Hering. * * * The mere operation does not cure strabismus; it is but a step in a very complex treatment, the aim of which is to reestablish the harmonious working of the movements of both eyes as required for perfect binocular vision. Operating for strabismus differs essentially from operating for, say, a cataract or a tumor. In comparison with a squint operation, we should call these operations *static* operations, whilst we should consider strabotomy as a *dynamic* operation. In fact, it does not aim at correcting an anatomical defect, 'putting a crooked eye straight,' but at reestablishing a *function*, and a most important function, one that distinguishes man from animals, viz., stereoscopic vision."

Viewing our task from this comprehensive standpoint, it must be evident that our "muscle snipper" must fall short of his mark, and that ocular muscle surgery demands as much scientific study and technical skill as is demanded of any branch of surgery. It must also be evident that to attain this perfection, *useful binocular vision*, which is the only safeguard against recurrence in these cases which must come to operation, we should operate early. We should operate as early as it can be determined that they are operative cases. Furthermore, if we must operate early, this means that we must operate on very young children under a general anesthetic. This, in turn, demands that we must have very definite methods of procedure, i.e., a predetermined mode of attack, a certain and definite means to correct a previously determined defect, as the child's eyes are usually relatively straight as soon as he is surgically anesthetized. This means accurate preoperative study and measurement of the amount of deviation under *differing degrees of stress at different distances,* and accurate operative methods to be carried out in the operating room.

Until we make a systematic and thorough analysis of our squint cases before they are brought to the operating room, and adopt very

definite operative methods, our squint surgery will be unsatisfactory and a reproach to our profession. No one is justified in doing a tenotomy because it is a relatively simple procedure, and because a few cases of squint are apparently cured by this method. As Landolt remarked above, squint surgery has for its object the *restoration of a function*, not the relief of a deformity, and this requires definite surgical procedures carried out early, before vicious habits are formed, before serious amblyopia occurs, and while it is still possible to develop binocular vision, or fusion, the only thing that will ever enable us to attain and maintain straight eyes.

Anesthesia.—The selection of the anesthetic to be used in a given case will depend largely upon the age of the patient. Ether anesthesia is the anesthetic of choice in all persons under fourteen to sixteen years of age. This is particularly true where a recession or advancement is to be done, and tenotomies should not be done on young children with the exception hereinafter to be mentioned. Ether should be given by means of an electrically driven force pump. A suitable tube with mouthpiece attachment should be used so as not to interfere with the toilet of the operation. An advancement, resection, or recession can best be done under ether in all children, and ether anesthesia is to be preferred in some grown subjects of a nervous temperament.

In grown subjects, a satisfactory anesthesia can be secured by the use of cocaine. After two or three instillations of a 5 per cent solution of cocaine, to which is added one drop to the dram of $\frac{1}{1000}$ solution of adrenalin, I usually massage the area over the insertion of the muscle to be operated upon with a 10 per cent solution of cocaine and adrenalin, using a very small pledget of cotton on an applicator. This massaging with the stronger solution makes it unnecessary to use any injection of either novocaine or cocaine, as is sometimes recommended. It is claimed that by saturating a pledget of cotton in the anesthetic solution and placing it over the area to be operated upon and holding it in place by closing the lids, is the most efficacious means of anesthetizing. Any injection will more or less disturb the field of operation and will make the dissection more difficult. Before beginning the operation, the eye is thoroughly irrigated to wash away any excess of the cocaine. At times it is advisable to drop into the opened capsule a drop of the 5 per cent solution of cocaine, if the anesthetic previously used is not entirely satisfactory.

When a local anesthetic is to be used, I invariably give a hypodermic of morphia. sulph. gr. $\frac{1}{6}$ to $\frac{1}{4}$, to which is added scopolamin $\frac{1}{150}$ gr. or $\frac{1}{100}$ gr., thirty minutes before the patient is brought to the operating room. This quiets the nerves, lessens the amount of local anesthetic required, and secures sleep after the operation, in the majority

of cases. In about 15 per cent there is some nausea following its use, but this is very seldom of any particular annoyance.

Condition of Patient.—As Jackson has well said, strabismus operations are not emergency operations, and should not be done until the patient is in the best possible condition. Where anemia is present, it should be corrected. Nervous, choreic children should be under the care of a competent neurologist, and the disorder corrected as far as possible. We are never justified in operating in the presence of any frank infection. Eyes have been lost from corneal suppuration[2] following tenotomy and advancement. Operative procedures should not be undertaken in the presence of acute colds, suppuration of the accessory nasal sinuses, lachrymal sac infections, blepharitis ulceroso, trachoma, or hordeolum.

When and on Whom to Operate.—This is such an important subject that it will be both interesting and instructive to review the opinions of some of the best known ophthalmologists, as expressed in ophthalmologic literature.

Javal,[3] in 1896, said:

"When operation must come to the aid of optical treatment, much tact is needed to choose the most propitious moment, late enough for the subject, prepared by the preliminary exercises, and arrived at an age when he can express his impressions, can attain all the desired effect, and still soon enough not to put off the moment of definite cure, and to reap the benefit of the courses of exercises instituted."

Frances Valk[4] says:

"Until a child is five years old, I tell the parents to let his eyes alone."

A. J. Ballantyne[5] says:

"I would not hesitate to operate at any age, having first, of course, ascertained from six months' trial of nonoperative treatment that a cure by these methods was unlikely to be obtained."

N. Bishop Harman[6] says:

"If the constant use of glasses that correct any error of refraction does not effect a rectification within a month, we may conclude that the glasses will not correct the squint. Any time allowed to elapse between this conclusion and operation, except so much as may be employed in attempting to regain the sight of the squinting eye, may be considered as lost time."

A. E. Davis[7] says:

"I believe it a good plan to operate as soon as the angle of squint ceases to improve with orthoptic measures. Nothing is to be gained by waiting, while much is to be lost. After surgical interference set-

ting the eyes straight exercises can be used to great advantage, often resulting in a complete cure.

"Age: rarely operate under two years of age. Orthoptic exercises suffice up to the age of three, then stereoscopic exercises for fusion training is followed by operation, if necessary. In constant unilateral squint that does not improve, and is accompanied by false fixation, operation should be done at two years or earlier."

Duane,[8] in 1910, said:

"Usually the longer we wait, the more unfavorable for operation the conditions become. Overtense muscles become rigid, overstretched muscles become enfeebled, and the ability of the eyes to perform fusion, or what is still more important, to overcome diplopia, becomes less."

Wendell Reber,[9] says:

"Other things being equal, we might well wish that every strabismic that comes for operation should be at least fourteen to sixteen years old. The operation then enjoys the following advantages: (a) the refractive side of the problem has in all probability been pretty well worked out; (b) the patient's cranium will have taken on more or less of its adult characteristics, although some outward sling of the long axes of the orbits frequently goes on up to thirty years of age."

Dr. Posey[10] in discussing Dr. Zentmayer's paper, said:

"That he made it a rule never to advance a muscle by the old methods in a child under ten years of age, on account of the difficulty and danger in introducing the needle into the sclera of less developed eyes."

Vard H. Hulen[11] says:

"In no case am I able to see serious objection to resort to surgery no matter how young the child, provided correct and complete non-surgical treatment has been previously given, and I am convinced that it is a highly desirable thing to obtain parallelism before the developing period for the eye has passed. We should not permit the parents of the child to postpone correct operative procedures indefinitely as so often occurs."

Josef Meller[12] does not approve of early operative interference in squint cases. He says:

"Therefore, in our clinic, we operate only on children who have passed their twelfth or thirteenth year."

That Dr. Meller still adheres to this practice is evident from the following taken from his article on squint in the February number of Wien. klin. Wchr., 1926, xxxix, p. 191.

"Even when the squint can no longer be influenced, still I would reject operation before the twelfth or fourteenth year, not only because sometimes in the course of time and growth the squint may decrease, or even disappear for some unknown reason, but also because

operation is not to be recommended until the child has become intelligent enough to allow of its being operated upon under local anesthesia. Squint operations under general anesthesia cannot be measured. A good cosmetic result can be obtained only with the patient's cooperation."

Dr. J. A. Kearney,[13] 1919, in describing his practice in the New York Polyclinic Medical School, advises as follows:

"Operations upon the external ocular muscles for the correction of squint are advisable when the patient is nine years of age or over, and all necessary nonoperative measures have been tried and have failed to correct the deformity."

Dr. Kearney advocates the operative correction of the esotropia in selected cases even where the squint is relieved by glasses.

Bishop Harman[14] says:

"But if we aim at a real cure, our object is to once more put the eyes into such a position that the brain can resume control over their movements and fuse the diverse images it receives from them. That is, we aim at the restoration of binocular vision. Looked at from this point of view, operation is not only reasonable, but necessary, and even urgent. For we do by operation permanently what the stereoscope can do temporarily. By the set of the stereoscope we can in effect temporarily correct the angle of the squint. By operation we undo the evil set of the muscles and so permanently correct the squint. In each case we can obtain conditions under which one image may be seen by each eye and carried to the fusion center. A fixed squint is like an obstacle to the brain. It is something it cannot overcome because of permanent changes set up in the muscles of the squinting eye."

It is my practice to operate as early as it can be determined that orthoptic treatment, together with glasses and atropin, is not going to correct the defect. This can certainly be determined in six months in the average case, and much sooner in most cases. If, after the use of glasses, atropin when indicated, use of pads to bring up lost visual acuity, and orthoptic exercises as indicated in the chapter on nonoperative treatment of squint, the degree of squint is not lessened, it is our duty to put the eyes as nearly straight as possible, and begin again with our orthoptic training. This must be done early to secure the aid of *fusion*, as *fusion training* is of little avail later. The more I treat crossed eyes, and the greater amount of experience I have, the more inclined I am to accept the teachings of the late Dr. Valk, who was of the opinion that practically *all* these cases are operative cases. He said:[15]

"It has been my observation that practically all these cases of so-called *cured* squint still have more or less esophoria, and many would

be relieved of the necessity of the constant use of glasses if they were relieved of the muscular unbalance by judicious operation."

There is no doubt but the use of the stereoscope to perfect binocular vision is more successful after operating in those cases with a decided defect. In fact, more can be done toward a permanent cure in a few minutes in the operating room than can be accomplished in weeks and even months of training with the stereoscope. If these eyes are set approximately parallel (and this can be done by means of proper study and measurement beforehand, and accurate operative procedure), the innate desire for fusion and the natural objection to diplopia will cause even the youngest child to acquire naturally binocular vision. This is the very reason we advocate early operative interference. If these children are permitted to become more or less amblyopic in one eye, the image from that eye is not so perfect as that from the good eye, and the tendency for fusion of the two images is not nearly so great. The earlier we operate, the less amblyopia from nonuse. The longer the eye remains crossed, the more complete is the suppression of the image of the deviating eye, and the more difficult it becomes to reawaken the diplopia, and, finally, to create a demand for fusion of the two images. Once the desire for fusion is well developed, if the eyes are set relatively straight, that desire will finish our work. Instead of having exercise of the fusion ten to twenty-five minutes at a time for two or three times a week, they naturally exercise that faculty every minute during the waking hours, and nothing short of a paralysis of an ocular muscle will break down this fusion.

I do not wish to disparage the beautiful results obtained by means of glasses, atropin, and orthoptic treatment, but wish to impress upon the reader the importance of securing an *early* correction in these squint cases. To wait until twelve to thirteen years to operate (Meller, Reber, et al.) means but one thing, a lost eye as far as its original function is concerned. No patient can be even approximately cured who is allowed to go with a monolateral crossed eye from three to four years of age up to twelve or thirteen years of age. Amblyopia will be present, suppression complete, muscular changes will have taken place, i.e., an hypertrophy of the internus and more or less relaxation or atrophy of the externus, and the desire for binocular vision entirely lost. The advocates of this "watchful waiting" method all admit that a real functional cure is impossible at this age and that they operate for cosmetic purposes only.

I feel that it is our duty to teach the profession and the public that any child at or above the age of five years who is cross-eyed is a neglected child, and should be relieved of its deformity. Furthermore, they should be taught that if glasses, atropin, and orthoptic

exercises do not relieve a case of squint in a few months, it is folly to expect glasses to ever relieve it. I have had many, many cases brought to me with the history of wearing glasses for years, having them changed every few months, and when told that glasses will never correct the deformity, they are much surprised, as they had been assured that eventually the eyes would become straight from the use of glasses only. It has been my experience that where atropin and glasses do not markedly reduce the degree of squint within the *first month,* they do not do so subsequently, even though they be used for years. This *glass delusion* should be banished from both the public and the profession.

Then let it be understood that we should operate as early as it can be determined that the patient cannot be cured otherwise, and that we should operate on all squint cases whose squint is not reduced within six months after proper treatment has been instituted.

Operations for Strabismus.—In Chapter I the History of Squint Surgery has been reviewed at some length. We shall now present some of the most popular operative procedures, all of which have been tried and found of sufficient merit to justify their presentation. Their particular indications, with their limitations, as observed in my practice, have been set forth.

I shall endeavor to present their advantages, indications, and contraindications without prejudice or bias, believing that the same end-result can be obtained by more than one method, and realizing that the perfect squint operation has not yet been devised. I shall describe more in detail those procedures which have been the most satisfactory in my hands, and shall recommend certain definite rules of procedure which I believe will be of benefit in ultimately bringing squint surgery to a more exact and scientific basis.

E. Jackson[16] has classified strabismus operations as follows:

(A) "Operations done to diminish the influence of a particular muscle or muscles upon the position and movements of the eyeball, as tenotomy, and operations to increase the effect of tenotomy.

(B) "Operations done to increase the influence of a particular muscle or muscles upon the position and movements of the eyeball, as operations for advancement, tendon-tucking.

(C) "Operations to modify the influence of a particular muscle or muscles so as to change the direction in which it will tend to turn the eye, or to alter the relative proportions of the influences exerted in producing different movements, as by lateral displacement of the insertions, tendon transplantations.

(D) "Miscellaneous operations to influence the position of the eyeball. Combinations of two or more of these operations may be done as a single operation."

For the convenience of the student, I shall discuss the operative procedures under the following headings:

(1) Tenotomy, complete and partial.
(2) Advancement, capsular and muscular.
(3) Resection (tenectomy).
(4) Tucking.
(5) Recession.

Tenotomy.—Complete tenotomy consists of cutting the tendon of an ocular muscle at its insertion into the globe. Myotomy consists in cutting the muscle farther back through its muscular fibers. A partial tenotomy consists of a partial detachment of the muscle from the globe by means of cutting the muscle tendon at one or more places.

The history of tenotomy has been given in Chapter I. There have been devised a number of methods of performing tenotomy. There are two types of tenotomy as practiced at the present day. They may be divided into the *open method* and the *subconjunctival method.* The open methods as practiced today are Arlt's and von Graefe's, or some modification of one or the other.

Arlt's operation is given by Knapp[17] as follows:

"The conjunctiva is grasped in front of the insertion of the tendon with fixing forceps (one branch up, the other down), raised in a fold, which is incised with squint scissors, so as to make an aperture of from six to seven millimeters. The branches of the forceps are now closed and moved two or three millimeters backward over the tendon, then opened seven to eight millimeters vertically to the sclerotic, to grasp and raise the tendon near its insertion. The tendon is detached with strabismus scissors from one end to the other as near to the sclerotic as possible. A small squint hook, introduced under the detached tendon, should pass unrestrained over the insertion. Arlt gives some very useful details in making this examination, which, being common to all methods of tenotomy, I shall here transcribe. The blunt (strabismus) hook should be used as a probe, introduced immediately behind the line of insertion of the tendon, its point held down upon the sclerotic, passed up just beyond the border of the tendon, and moved towards the cornea. If it is stopped, some fibers have been left, which must be cut. The same is done downward. Pushing back the hook too far upward engages it in the muscle, and the cellular and vascular tissue, without finding the stray fibers.

"The wound is closed with sutures when by examination the effect is found satisfactory."

Of this most careful operation, Beard[18] makes the following interesting remarks:

"Von Arlt, of Vienna, made tenotomy after the manner of v. Graefe, save that he picked up the exposed tendon with the mouse-tooth or toothed fixation forceps, instead of with the hook, but used the hook

in finding and severing remaining fibers. This surgeon was most circumspect in determining the position and extent of his incision through the membranes, choosing a small horizontal one over the center of the tendon for the lower degrees of strabismus and a freer vertical one over the insertion for the higher grades. If it was feared that the separation of the muscle from the globe was extreme, the aponeurosis of the tendon was included by the suture which finally closed the vertical wound; if not, only the conjunctiva was included."

It will be observed that practically this same procedure is recommended by J. Meller, of Vienna, as shown in Fig. 73.

Von Graefe's Method.—A vertical incision is made in the conjunctiva about seven or eight millimeters long between the cornea and the insertion of the tendon. The dissection is continued downward toward the canthus, beyond the tendon's insertion in the muscle. A strabismus hook is introduced underneath the tendon now, and traction made on it sufficiently to bring the tendon well into view, and with a pair of scissors the tendon is completely detached from the eyeball, cutting close to the sclera. A hook is now introduced to search for any uncut fibers of the tendon. This will not be necessary if a good exposure of the tendon is secured before cutting the tendon. The conjunctival wound is now closed with fine sutures. This method, with very slight modifications, is much used at the present time.

Subconjunctival Tenotomy.—This method of tenotomy was first introduced by Jules Guerin and Baynen (Knapp), but was perfected by Critchett, of London,[19] and is usually spoken of as Critchett's operation. Beard[20] describes Critchett's operation as follows:

"With strong mouse-tooth forceps, pressed firmly upon the globe over the lower border of the tendon, just behind its insertion, a horizontal fold of conjunctiva and underlying capsule of Tenon is picked up and cut crosswise with blunt-pointed scissors close to the forceps, if possible dividing both membranes at one snip, though it may be necessary to pick up the capsule in a similar fold and incise it separately. Thus a vertical wound opening is made. The lower border of the tendon is now brought to view by gently lifting with the forceps the fold of membrane still retained in its jaws and touching the wound with the sharpened point of a cotton sponge. A delicate, flattened Graefe hook is then inserted, point up, beneath the tendon, pushing it snug up to include all the fibers, the forceps relinquished by the left hand for the hook, which is slightly raised. One point of the delicate blunt-pointed scissors is passed beneath the tendon close to its attachment and the other beneath the conjunctiva, and the tenotomy accomplished by successive snips of the scissors, cutting from the heel to the point of the hook. As in the Graefe method, stray fibers that have escaped the scissors are sought and divided. If still greater effect is desired, a subconjunctival incision is made vertically in Tenon's capsule, a little back of the point where the tendon was cut. Unless there is a tendency of the conjunctival wound to gape, no suture is employed."

Snellen's Operation.—Snellen[21] of Utrecht, devised a very ingenious tenotomy operation which consisted of making an incision directly through the conjunctiva and central part of the tendon at or near its insertion. Through this opening he inserts a small strabismus hook, point upward, and incises the remaining upper portion of the tendon with a pair of blunt-pointed scissors. The hook is now turned point downward, and the lower portion of the tendon is picked up and severed likewise, after which a suture is placed in the conjunctiva if found necessary.

Dr. George T. Stevens, of New York, modified Snellen's operation, and devised the most exact and efficient instruments for partial tenotomy. In Stevens' operation the upper and lower margins of the tendon are not severed.

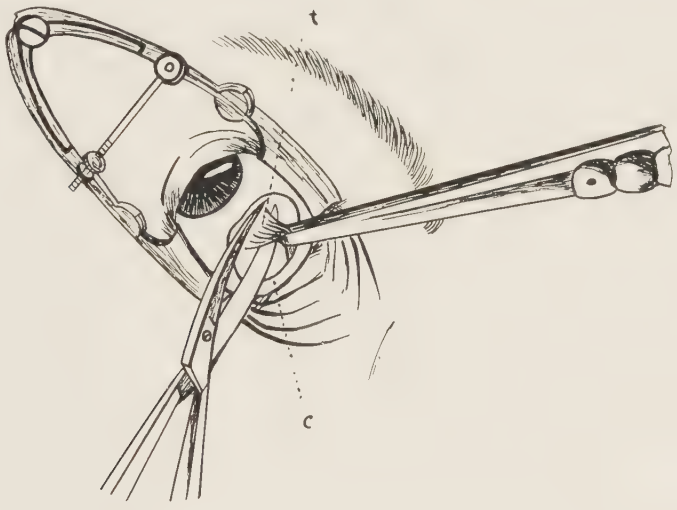

Fig. 73.—Tenotomy of the rectus internus. The eye is placed in the position of abduction. A small incision is made in the conjunctiva (*c*). The forceps, after seizing the muscle, are held perpendicular to the curvature of the eyeball. One blade of the open scissors is introduced immediately behind the muscle at its insertion, the other blade lies in front of it. A single snip with the scissors severs the tendon (*t*) at its insertion. (From Josef Meller, *Ophthalmic Surgery.*)

The following very interesting operation for tenotomy, a very slight modification of Von Arlt's is given by J. Meller[22] as shown in Fig. 73.

"The operator, with a pair of toothed forceps in the left hand, raises a fold of bulbar conjunctiva at a distance of three mm. from the limbus, and with slightly curved scissors makes a vertical incision, from five to seven mm. long. The nasal border of the wound is then lifted up, the subconjunctival tissue severed and undermined by short cuts, and the closed forceps, held parallel to the sclera, introduced until they reach the insertion of the muscle (five mm. from the limbus). They are then opened, pressed against the sclera, and the tendon is grasped. The forceps are now rotated into a position perpendicular to the surface of the eyeball, so that the tendon, covered by Tenon's capsule,

is somewhat raised from the sclera. The insertion of the tendon is seen as it glistens through the capsule. The scissors, held in the right hand, make a short incision at the lower margin of the insertion of the tendon, which at the same time opens Tenon's capsule. Through this gap one blade of the scissors is pushed behind the tendon, and the other in front of the insertion. The convexity of the blades is directed toward the sclera, so that the points are therefore turned away from the eyeball. In this position of the scissors no injury of the sclera can occur. If the tendon is properly severed, only a slight ridge remains on the sclera marking the point of the tendinous insertion. The incision includes only the width of the insertion of the tendon, and does not touch the attachments of Tenon's capsule to the eyeball beyond."

Tenotomy of External Rectus.—This is indicated in slight degrees of divergence and in higher degrees of divergence when accompanied with a resection or advancement. The effect of a tenotomy of the external rectus is not so great as that of the internus. Its execution is the same as tenotomy of the internus, with the exception that it must be remembered that its insertion is farther from the cornea. One need not fear the bad after effect which sometimes accompanies a tenotomy of the internus. It has been my observation that about ten to twelve degrees of deviation is corrected by a tenotomy of this muscle. Knapp says it varies from fifteen degrees down to no correction whatever. Wootten[23] has given us definite rules for our guidance in tenotomy of the external rectus. (See Divergent Strabismus, p. 214.)

Tenotomy of the Superior Rectus.—A correction of approximately seven to ten degrees is usually obtained by a tenotomy of this muscle. Concerning the tenotomy of this muscle, E. Landolt says:[24]

"Tenotomy of the superior rectus muscle produces an alteration of about five degrees, but it is quite impossible to predict the exact effect of any advancement, tenotomy, or shortening, which may be necessary in any given case."

It is a justifiable procedure where there is a hypertropia of eight degrees or more in a person whose occupation does not demand a great deal of close work. In such a case, in a person whose trade or temperament demands much close work, it would probably be advisable to advance the antagonist. A tenotomy of this muscle should be done with the greatest care and delicacy, as the end-results are not so certain as those of the lateral muscles.

Tenotomy of the Inferior Rectus.—I only mention this to warn the reader against its performance. I personally performed this operation but once, and had cause to regret it afterwards. I never expect to have to regret the second offense. The reason for trouble here is that the demand on the inferior rectus is so great in our daily life, the eye

being turned down while walking, eating, or reading; to cripple it means trouble. An advancement of the antagonist is always indicated.

Tenotomy of the Obliques.—In congenital or acquired squints, with a decided deviation upward and outward, a tenotomy of the inferior oblique is indicated. This operation was particularly advocated by Duane,[25] who describes the operation as follows:

"Tenotomy of the *inferior oblique* is done by dividing the tendon of origin of the muscle through a cutaneous incision 15-20 mm. long, made at the lower inner angle of the eye just below the lachrymal sac. The incision is carried down to the bone, and the orbital septum is divided just where it joins the lower and inner margin of the orbit. A squint hook is swept inward along the floor of the orbit with its point directed upward and inward until it engages the tendon. The latter, which is more or less buried in fat and fascia, is completely divided with the scissors. The tendon usually retracts, but to prevent possible readhesion two or three mm. are exsected. The effect is a complete paralysis of the muscle."

This tenotomy can be done through a conjunctival incision (C. Berens).

Posey[26] reports a number of cases and gives indications for, and results of, this procedure. This operation is sometimes indicated, even on very young children (Duane).

A tenotomy of the superior oblique can be made by making an incision into the brow over region of the trochlea. Its indications are not so frequent or so definite as are those for a tenotomy of the inferior oblique.

Tenotomy: Its Indications and Contraindications.– It may be permissible to do a tenotomy of the internal rectus on grown subjects with marked deviations and marked amblyopia where there is serious objection to any operative procedure on the good eye. It would be more scientific and accurate to do a recession of the internus and a definite advancement of the externus. (See Case 10 in chapter of Illustrative Cases.)

Contraindications.—An internal rectus tenotomy is never justifiable in a patient under twelve years of age. No one can tell what the result of a tenotomy is going to be, and it is certain that a tenotomy of an internal rectus interferes with convergence; hence with the use of the two eyes for close work; that means tenotomy is never justifiable where there is any hope of securing binocular vision. As a matter of fact, I believe that tenotomy will grow less and less in favor, owing to its indefinite result and also owing to its bad end-result; i.e., causing late divergence in a certain number of cases. I believe that a certain definite recession with or without advancement will eventually take the place of tenotomy. I have recently combined the two with much accuracy and good results (see Case 6), and I believe these

accurate methods will supersede the inaccurate and indefinite ones, much to the benefit of the public and the credit of ophthalmic surgery. Tenotomy of the external rectus is indicated where there is an excess of divergence associated with external squint, and I have seen no harm come from this operation; however, it has been my experience that single tenotomies of this muscle without an accompanying advancement have but little effect. Associating the recession operation with advancement in divergent squint, I find that recession is not particularly indicated here, as a free tenotomy has no disadvantages and causes much less trauma than a recession. Tenotomy of an oblique is indicated where it can be shown that there is a decided overaction of the muscle.

Duane[27] says that the most important indication for tenotomy of an inferior oblique arises where there is a paralysis of a superior rectus associated with a secondary deviation caused by overaction of the inferior oblique in the opposite eye.

The author has discussed tenotomy at more length than its real value as a surgical procedure at the present day deserves, but inasmuch as it is being, and will be, frequently performed, he feels that its virtues and pitfalls should be fully discussed. In the future of squint surgery I believe that tenotomy without *some* restraint or control will seldom be performed. This I believe will come about for two reasons. In the first place, I feel that when the public and the physicians are better informed, the majority of squint cases will be operated on so early in life that a tenotomy will not be needed or in any way indicated, but, on the contrary, contraindicated. In the second place, in those older subjects, in whom there is some excuse for a tenotomy, the more exact procedures of recession and advancement or resection will take its place.

Complications.—It must be conceded that tenotomy is one of the safest and easiest operations done upon the eye. This very fact has made it popular, particularly with the timid patient and with the timid operator, if there be such. But it is a mistake to feel that no harm can come from a tenotomy. Knapp (Norris and Oliver) reports the loss of an eye. H. Derby[28] reports two cases of perforation of the sclera. Massive hemorrhages have occurred. These more severe complications are rarely seen, but they must not be forgotten. Some of the lesser complications are: Tenotomy of the muscle of the wrong eye. This could scarcely be called a misfortune, as a tenotomy of the straight eye is advocated by some writers.

Bettremieux[29] says:

"For five years I have maintained the theory that the part of the squint deviation which relieves the trouble of innervation of convergence is accomplished by an operation on the eye which does not squint."

Parinaud[30] advocates the tenotomy of the sound eye. The tenotomy of the wrong muscle on either eye would be a grave accident, and one which would require immediate attention.

The majority of our troubles following tenotomy of the internus are due to a too-free tenotomy. These are retraction of the caruncle; loss of motility, particularly loss of converging power; exophthalmus, with widening of the palpebral fissure, causing the eye to look larger; and, lastly, the much-dreaded divergence, which produces more deformity than was present before the operation.

Methods for Controlling Tenotomy.—Knapp[31] mentions under heading of procedures to diminish the effect, *application of sutures.* He recommends a scleral stitch, which also includes the muscle tendon. I can indorse this stitch as being efficacious. The effect of the tenotomy can practically be nullified by this stitch, when properly placed and securely tied. A lesser effect can be obtained by catching only the capsule and conjunctiva. Beard (l.c.) advises immediate control of the effect of the tenotomy where it is evident that too great an effect is produced. He advises this, even at the risk of reestablishing the squint, as he considers the internal squint thus left easier to correct than the divergent squint which would follow. Good effects can be obtained by watching the case daily and removing the central suture at the proper time. If the central suture is causing convergence, it should be removed the next day.

Procedures to Increase the Operative Effect.—The older writers recommended the extension of the wound upward and downward, thus cutting more the capsule and the check ligaments. This is the chief cause of the after divergence which has just been discussed, and this procedure is never justified. Many sutures have been applied (Knapp, von Graefe, Jocqs) to anchor the eye, to aid in controlling the antagonist, etc., but most of them are ineffectual. If one wishes a real aid to a tenotomy, some form of shortening of the antagonist (see Advancements or Resections) should be performed, and the methods of exactness, hereafter to be considered, should be adhered to. This procedure is particularly indicated in cases of divergence.

Partial Tenotomies.—Partial tenotomies may be divided into three classes. Those in which the central portion of the tendon is cut, those in which its lateral portion is cut, and, third, a combination of the two.

Knapp, on remarking to von Graefe that he was doing marginal tenotomies, receives the curt reply: "You will not do that long."[32]

Von Arlt[33] says of marginal tenotomies:

"If I made a tenotomy for simple strabismus, or for insufficiency, and, afraid of an overcorrection, left only the one or the other fiber undivided, the effect was only transitory."

Knapp soon abandoned partial tenotomies for more definite measures.

Beard[34] recommended the cutting of the central portion of the tendon, and the advancement of the externus in cases of squint, a very good procedure.

Savage[35] says:

"Yet it will be shown in this section that partial, and not complete, tenotomies, only, should be done, even in higher degrees of heterotropia."

On page 8114 he says:

"The ideal operation for the cure of esotropia and its complications consists of advancements of both externi, to make them stronger, and partial tenotomies of both interni to make them weaker."

The above advice is particularly sound, when one takes into account the exact degree of deviation and the exact amount of shortening of the externi.

To Dr. George T. Stevens, of New York, we are indebted for the most exact and painstaking partial tenotomies. He often operated a number of times before securing the desired effect.

Stevens[36] describes his partial tenotomy as follows:

"The surgeon takes with his fine forceps a minute fold of conjunctiva at the center of the insertion of the tendon. Drawing the little fold of conjunctiva slightly away from the eyeball, with the extreme points of his tenotomy scissors (Fig. 136) he snips the fold transversely so that an opening about half a millimeter in extent is made through the membrane. Now the forceps, the points being closed, are pressed into the little opening and slightly backward, when the points are permitted to spring apart, after which they are again closed, this time holding a small fold of the tendon just behind the insertion. This little fold of tendon being put on the stretch, the fine pointed scissors, by little snips, dissect the tendon from the eyeball between the layers of the capsule (which should remain intact) toward one border of the insertion. The sense of feeling of the fingers against the rings of the scissors will, in the hands of one skilled in the operation, inform him of the approach to the border and warn him against its destruction."

It must be evident to the reader that such exact surgery requires unusual skill and a very large experience, together with the use of such matchlessly perfected instruments as those introduced by this master. I feel certain that the average operator will succeed better and do less harm if he makes a more extensive exposure of his tendon. A slightly greater scarring of the conjunctiva over the tendon is far outweighed by the greater exactness secured by more exposure.

Verhoeff[37] has introduced a very ingenious method of partial tenotomy, as shown in Fig. 74.

F. C. Todd[38] recommended the partial cutting through of the tendon of the internal rectus. He made two cuts in the tendon on one side and one cut on the opposite side, each cutting to its center, the cuts being about five millimeters apart and not opposite. (Fig. 75.)

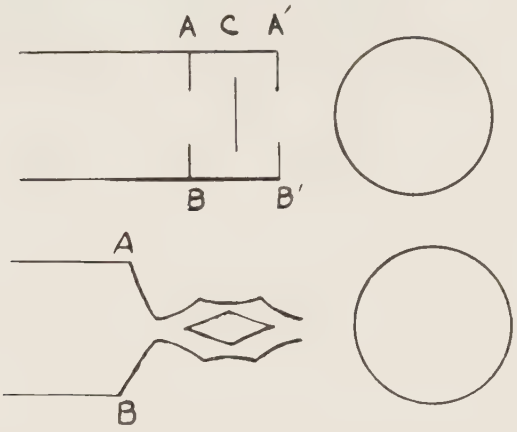

Fig. 74.—Partial plastic tenotomy of Verhoeff. Diagram above shows the points *A B, A′ B′,* and *C* at which the tendon is partially divided. The diagram below shows how the tendon becomes lengthened by giving way after the operation. (*American Encyclopedia of Ophthalmology.*)

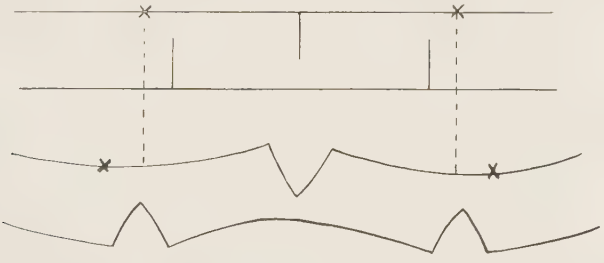

Fig. 75.—Diagram of Todd's method of limited tenotomy. The change in location of crosses shows the lengthening which takes place. (From Todd, *Journal of the American Medical Association.*)

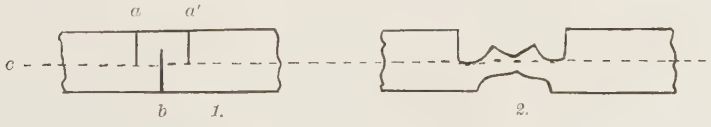

Fig. 76.—Harman's partial tenotomy. 1. Shows *aa′* cuts on one side of tendon to middle line *c*; *b* intermediate cut, 2/3 width of the tendon.

2. Tissue stretched after cuts are made showing band uniting axis of tendon. (Drawn from effects obtained by cutting strapping.) (From Harman, *Ophthalmoscope.*)

N. Bishop Harman,[39] London, has described a partial tenotomy operation, which is effective and when properly performed is *safe*. It is probably more effective than either Todd's or Verhoeff's.

Fig. 76 is sufficiently descriptive of Harman's operation.

I have tried this operation, and have found it safe and effectual. I have used this method of weakening those so-called "abnormally short" or at least abnormally strong muscles as indicated by the pull upon the scales. (See Fig. 92.) By this means a muscle whose pull is 125 grams can be reduced to 75 grams, and the accurate results can be obtained by certain definite degree of resection. (See Case 15, chapter XII.)

Duane (40) says:

"The central buttonhole operation produces no material effect unless the tenotomy is total; i.e., severs the whole insertion."

It is his opinion that the only way to obtain graduated effect is by carefully dividing the lateral attachments until the desired result is obtained as indicated by repeated tests. In my earlier practice I tried the central partial tenotomies, but found these of no particular value; however, I feel that they may have some place in squint surgery when used as recommended by Beard and Savage. The only objection to their use would be the uncertainty as to what their ultimate effect, if any, would be. I see no reason why this procedure would not be efficacious if its effect were regulated by means of the scales as above indicated.

Advancement Operations.—By this term is meant the bringing forward of the tendon of the ocular muscle and attaching it to the globe nearer the cornea, thus giving it greater power over the movements of the eye.

The first advancement operation was performed by Guerin, who, in an effort to correct a divergent strabismus following a double tenotomy of the interni, picked up the remains of the internus and brought it forward and attached it to the sclera (Jackson). In the Medical Times and Gazette, 1857 and 1858[41] is to be found the description of Critchett's advancement. This was a three-thread advancement operation with a definite effort at bringing the tendinous insertion nearer the cornea. (See Fig. 4, p. 25.)

In 1871, Weber[42] described a single-suture advancement. De Wecker[43] in 1873 described a similar operation, which seems to have been known as de Wecker's advancement.

The number of advancement operations introduced since these have been legion, all of which have been based on more or less the same principles. Among these may be mentioned Agnew's;[44] Landolt's;[45] Prince's;[46] William's;[47] Beard's;[48] Howe's;[49] Argyll;[50] Melville Black;[51] Wootten;[52] C. Worth;[53] Verhoeff;[54] Valude's[55] splitting tendon advancement.

It will be seen from the above very incomplete list that it would be futile to undertake the description of so many operations. The author

will describe a few of the most important, selecting some for their historical or inherent worth, pointing out the good and weak points as have occurred to him in their use.

Critchett's advancement was improved upon by Jessop[56] by tying his suture into the tendon. H. D. Noyes[57] also improved upon Critchett's operation by using a very fine, sharp needle by which he could more easily secure a good scleral hold. This is practically the same advancement operation described by J. Meller.[58]

The following very simple and efficient advancement operation, described by E. Jackson[59] is well worthy of mention. Jackson especially recommends this operation for the relief of divergent squint. The author has found it very effectual.

"A curved incision in the conjunctiva and episcleral tissue is made 10 mm. long and concave to the cornea. The flap toward the canthus is held up and dissected free from the sclera by snips of the scissors. The dissection is first to be made a little above or below the insertion of the internus, until one blade of the Prince advancement forceps can be slipped beneath the tendon, back from the insertion almost as far as it will be necessary to place the suture. The other blade of the forceps is pressed on the surface of the conjunctiva, so that the whole mass of tissue to be advanced is caught between the blades, which are closed upon it. With the flap thus held the insertion of the tendon, and all other adhesions of the flap to the globe, are divided by snips of the scissors. The flap can then be drawn forward into its desired relation with the eyeball and the position of the suture necessary to retain it there, and the amount of tissue to be removed, decided on.

"The finest, sharpest curved needle is then passed through the flap from the conjunctiva to the scleral surface, back of the blades of the advancement forceps and about 2 mm. above (or below) the center of the corneal margin and 1 mm. from it, in such a way as to take a firm hold in the sclera without passing through it. It should include one-fourth or one-third the thickness of the sclera; and the points of entrance and emergence should be 3 to 4 mm. apart. If, at the first attempt, the needle cuts or pulls out of the firm tissue, it should be introduced a little deeper and a little farther back from the cornea. The needle is then passed beneath the flap and through it from the sclera to the conjunctival surface, back of the blades of the forceps, opposite the original point of entry, and 2 mm. below (or above) the center of the tendon; that is 4 mm. from the first entrance. (See Figs. 77 and 78.) The flap should be drawn forward and the suture tied. If there remains redundant tissue this may be trimmed away, generally including that squeezed by the forceps."

Jackson recommends that lateral sutures be placed when found necessary to flatten out the muscle on the globe or to perfect the conjunctival closure.

Single suture operations are not usually satisfactory, as they tend to bunch up the tissues, distort the normal anatomic relations, and are inclined to make pointed attachments.

Worth[60] describes his operation as follows:

"Each needle and suture before being inserted is drawn through a fold of lint on which is a little sterile vaseline. One of the needles is passed inward at *A′* through the conjunctiva, and brought out at *B′*. The bight of the thread thus encloses a width of about 2 mm. near the upper edge of the muscle, together with capsule and conjunctiva. The ends of the thread from *A′* to *B′* are then crossed over, making a half-hitch at *C*. The end bearing the needle is then entered at *D* and passed through conjunctiva, capsule, and muscle, and carried beneath the lower blade of the advancement forceps, out of the wound, and stuck into a piece of gauze which has been placed on the patient's forehead to receive

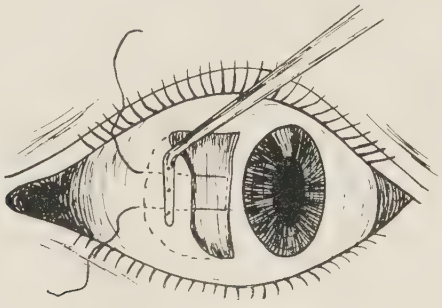

Fig. 77.—Jackson's advancement with single scleral stitch. Tissue held by forceps and scleral stitch placed ready to tighten, and later cut away tissues at the broken line. (*American Encyclopedia of Ophthalmology*).

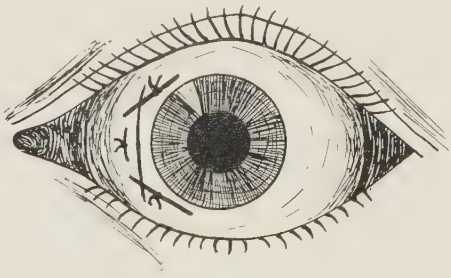

Fig. 78.—Jackson's advancement with scleral stitch in center, and superficial stitches above and below, at completion of operation. (*American Encyclopedia of Ophthalmology.*)

it. The suture *A*, *B*, etc., at the lower margin of the muscle is then similarly dealt with, its needle also being brought out of the wound and stuck into the gauze.

"The anterior parts of the muscle and capsule and conjunctiva are then removed by cutting them through behind where they are grasped by the advancement forceps. The longitudinal position on the muscle of loops, *A*, *B*, *C*, and *A′*, *B′*, *C′*, and the amount of tissue removed, vary according to the degree of rotation required. If only a very small effect is required perhaps no tissue at all may be removed. The sutures on the muscle are placed 2 or 3 mm. behind the cut end, in order that there shall be no tension on the extreme end of the muscle while union is taking place.

"The next stage in the operation, the insertion of the two main sutures into the sclerotic at *G* and *G'* is one that requires great judgment and delicacy of touch. Take one of the needles in the holder, leaving about ¼ inch exposed. With the fixation forceps take a firm hold of the globe at the site of the old insertion. Lay the back of the needle on the sclerotic exactly in line with the position of the suture in the muscle (in other words, keep the suture parallel with the marking suture) and about ⅛ inch or a little more from the corneal margin. Press the point of the needle backwards slightly, so as to dimple the sclerotic a little. Then push it onward so that it traverses at least half the thickness of the sclerotic, but taking the greatest care not to pierce the whole thickness. The superficial extent of the insertion of the suture is about ⅛ inch. A fresh hold of the needle is now taken with the holder just below the point and pulled through. The other main suture is similarly dealt with. No verbal directions can be given for gauging

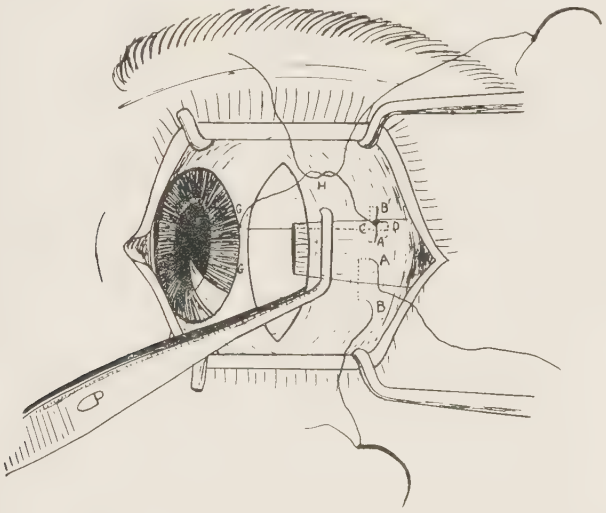

Fig. 79.—Worth's advancement operation. (From Worth, *Squint.*)

the depth. One judges by sight and touch. One's tactile sense also warns one of an abnormal thinness of the sclerotic, which is occasionally, though rarely, met with.

"I now use a split-eyed needle with cutting edges for inserting the sutures into the sclerotic, but consideration of this is postponed to a later page in order to avoid complicating the description of the operation.

"The assistant grasps the eyeball with fixation forceps on the opposite side, and rotates it into the primary position. The surgeon ties each suture at *H* and *H'*, with the first hitch of 'the surgeon's knot,' gradually tightening it as he manipulates the anterior end of the muscle with forceps into the desired place. Care must be taken that the anterior end of the muscle be neither inverted nor everted. The assistant now lets go his hold of the eye. The tension of the sutures is adjusted and each knot completed by a second hitch. These two sutures bear all the weight. The marking sutures are now tied. If there be any gaps in

the membranes above or below the holding sutures, they are closed with additional fine sutures.

"In operating under cocaine, before the knots are tied at *H* and *H'*, the assistant holds the globe in the primary position with the forceps, while the patient is told to try to look away from the operated muscle. This relaxes the muscle while it is being drawn forward by the sutures. The sutures having been temporarily secured at *H* and *H'* by the first hitch of 'the surgeon's knot,' the assistant releases the globe. The fine adjustment is done by tightening or loosening the hitches at *H* and *H'*, the result being checked by the mirror test or by the reflection of a candle flame on the cornea. The knots at *H* and *H'* are then completed.

"The immediate effect is the permanent result. No overcorrection, therefore, is necessary." (Fig. 79.)

The author has not found the immediate effect of any advancement *by any method* to be the permanent effect, when advancement alone is done. If some means is not used to prevent the constant drawing of the

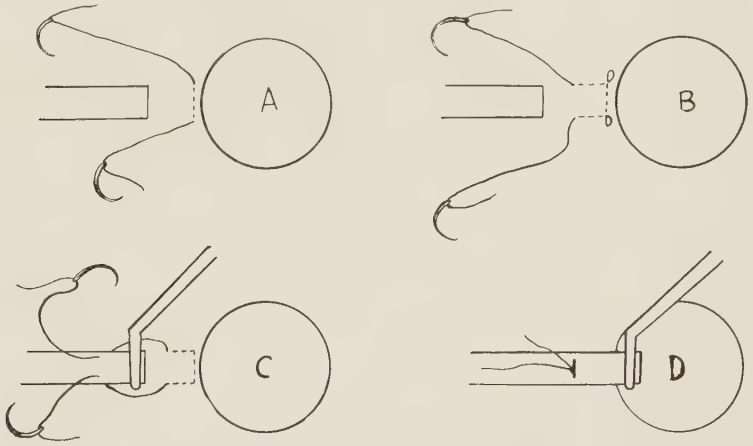

Fig. 80.—Advancement stitch of Verhoeff showing different stages. The dotted lines indicate the part buried in the sclera. (*American Encyclopedia of Ophthalmology.*)

internal rectus on the sutures placed in the advancement of the external rectus in cases of comitant convergent squint, the immediate effect will always be greater than the final effect. This does not necessarily obtain where a tenotomy, recession, or even a partial tenotomy, has been done.

Verhoeff[61] has given us one of the best advancement operations. This is described as follows by Jackson:[62]

"The tendon is exposed through a vertical incision 3.5 mm. from the cornea, raised on a strabismus hook and isolated. It is then seized with the advancement forceps near the insertion. A double-armed silk suture is used. It is first introduced into the sclera for 2.5 mm. as shown in *A*, one-half to one mm. from the corneal margin. Then each needle is introduced at the point of exit of the suture, and carried horizontally 2.5 mm. towards the insertion of the tendon as shown in *B*. The tendon is then divided at its insertion and the two needles passed through it

sufficiently far back, *C*. The tendon is then brought forward with the forceps, the eyeball rotated into position, and the suture tightened, *D*. A sufficient portion of the tendon grasped in the forceps is excised and the conjunctiva brought together by two superficial sutures.'' (Fig. 80.)

I have performed this operation a number of times, and have found it very accurate, safe, and effective. I can recommend it particularly in divergent squint, when associated with a tenotomy of the antagonist.

The following interesting operation of Dr. W. B. Lancaster deserves a place in squint surgery. Its author claims to be able to adjust the amount of shortening done with more accuracy than with the Reese, Verhoeff, or Jackson, operations. (Figs. 81 and 82.)

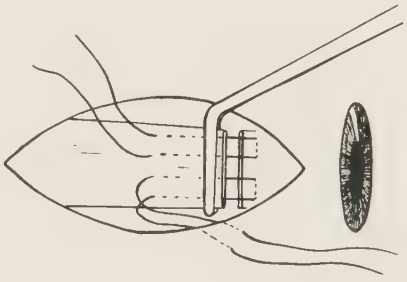

Fig. 81.—Dr. Lancaster's latest method of operating.

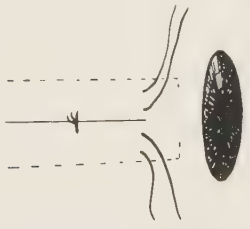

Fig. 82.—Dr. Lancaster's latest method of operating.

''The conjunctival incision is parallel to the limbus about 8 mm. from the cornea, that is, slightly posterior to the insertion of the muscle. The reason for this is that the stitches which come up through the stump or insertion of the tendon, through the belly of the muscle and through the conjunctiva, may be in the anterior conjunctival flap and not in the posterior. Result, a better scar—no puckering. Length of incision about 10 mm. Undermine the conjunctiva forward to the limbus, also undermine backward exposing the capsule of Tenon over the muscle. Pick up the capsule with forceps at the margin of the muscle and buttonhole it, permitting the insertion of the muscle hook under the muscle. Isolate the muscle by cutting the capsule along both edges, so that the muscle can be raised on two hooks.

''Apply the Prince's forceps to the muscle at the proper point. This point is one to two millimeters in front of the future insertion

of the muscle. For example, if you wish to shorten the muscle 8 mm., apply the forceps 6 or 7 mm. from the original insertion. Then the stitches will be 8 mm. from the original insertion. The forceps are to be applied so that the handle is back over the muscle to be advanced, not forward over the cornea as is more usual. The reason for this is that the muscle can be pushed forward under the conjunctival flap toward the limbus more easily when the handles are in this position; there is less tendency to wrinkle the conjunctival flap.

"Having applied the Prince's forceps, cut the tendon not too close to the insertion, so as to leave a little stump.

"If there is much muscle hanging from the forceps, cut off the surplus, leaving one millimeter only, so that there will be no excess under the conjunctival flap to make a bunch. However, the excess will flatten down and make no permanent hump, even if it is left.

"Insert the two double-armed sutures, one through the upper third and one through the lower third of the tendon stump. Pass each needle from behind forward, emerging a little in front of the old insertion. Then pass the two sutures through the muscle a little back of the Prince's forceps. The needles enter on the scleral surface, and come out on the conjunctival surface. Next pass the two sutures through the anterior conjunctival flap. They must be placed in the conjunctiva directly over the points where they came through the tendon stump, so that the conjunctiva will be tied down on to the insertion in its normal position without puckering.

"The assistant fixes the eyeball with forceps applied on the opposite side of the cornea. Holding these in one hand, he, with the other hand, pushes the muscle held by the Prince's forceps under the conjunctiva toward the limbus, while the surgeon by gentle traction pulls the sutures into place and then ties them firmly. Thus the muscle is firmly held by two mattress sutures against the old insertion, where it becomes reattached. After the knots are tied, the Prince's forceps are released and gently extricated from under the flap. The knots are outside the conjunctiva, and so the sutures can be removed after seven or eight days. If 20-day catgut is used, the stitches can be left, or the knots can be cut off if they are too long in absorbing.

"The opening in the conjunctiva closes without suturing, if the posterior flap was not too freely undermined."

Dr. P. C. Jameson's operation for old paralysis of external rectus with extreme adduction, as described by him is as follows:

"The procedure consists of an extreme recession and an advancement after exsection as far back as external canthus of external rectus capsule and conjunctiva to sclerocorneal margin.

"The object obtained is to anchor the rim of outer canthus to corneal margin.

"The recession is done in the manner described by the author in previous writings. It violates the general rule by scleral fixation of the muscle end behind the equator.

"The dissection for the advancement and exsection of externus is a gross one, the muscle capsule and conjunctiva are not separated but a flap with these structures in normal continuity is dissected back to the fornix behind outer canthus.

"The line of exsection is behind the outer canthus.

"After this is accomplished a quadrant of conjunctiva is removed between the muscle stump and the corneal margin to which the outer canthus, muscle capsule and conjunctiva are sutured.

"These single sutures or two single and one double armed (if a central loop is preferred) are passed first through the inter marginal space (the edge) of outer canthus then through flap, conjunctival capsule muscle and on to sclero corneal margin where they are drawn taut and tied.

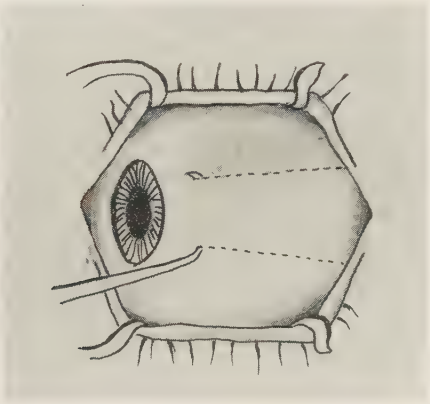

Fig. 83.—Outline of flap; hook introduced. (Dr. Jameson's operation.)

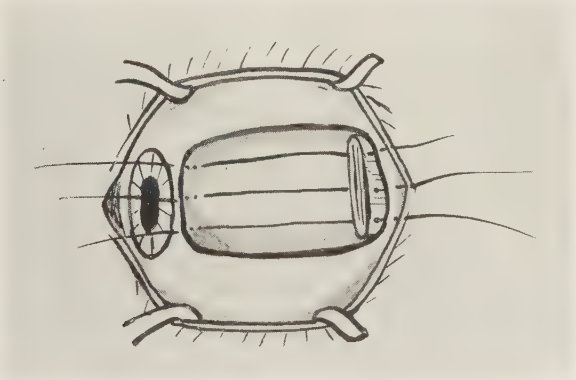

Fig. 84.—Muscle detached; flap exscted, sutures in position. (Dr. Jameson's operation.)

"If support to advancement is desired two loop sutures may be introduced—one to the upper, the other to edge of muscle insertions of the opposing side (internal rectus) and anchored respectively to upper and lower lid margin as far to the outer parts as will not over-ride the cornea. They pull the eye outward toward the advanced parts and are released and removed after they have served their purpose.

"The immediate result is to adduct the eye so that the outer canthus is brought up to the corneal margin which, of course, is an overcorrection but as the connective tissue band stretches, the eye is maintained

in position near the median line. Of course no muscle action is obtainable.''

Figs. 83 and 84 represent Dr. Jameson's advancement operation.

Resection Operations.—Under this heading is to be classed those operations in which the muscle is shortened and attached to or near its original attachment. Most writers (Jackson, E. C. Ellett, et al.) maintain that in all advancements the real attachment of the muscle is not near the cornea, but over the site of the original tendinous insertion. (This has been disputed by Worth, Landolt, Wells, etc.)

One of the first resection operations was that of J. F. Noyes,[63] in which (see Fig. 85) he shortened the muscle by placing a double-armed

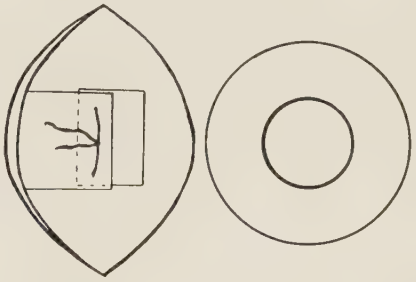

Fig. 85.—J. F. Noyes' shortening. (From Beard, *Ophthalmic Surgery.*)

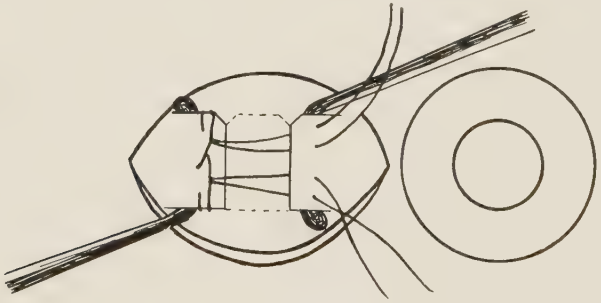

Fig. 86.—Schweigger's resection operation for squint. (From Beard, *Ophthalmic Surgery.*)

suture through the stump of the tendon and the muscle. Noyes did not cut off any of the muscle, but caused a shortening of it by overlapping.

Blanco[64] places the muscular end of the tendon underneath the tendinous stump. This method was used by Noyes according to the drawings. (See Reese resection.)

Callan[65] has removed as much as 12 mm. from the external rectus and claimed to have corrected as much as 35 degrees of deviation by so doing. This has not been my experience. As a rule, only about 22 to 24 degrees of correction can be secured from that amount of resection, when a resection only is done.

One of the best resections is that of Schweigger[66] and is described by Beard,[67] at some length.

Fig. 86 is self-explanatory.

It will be observed how very similar this operation is to that of Reese, which is the best known resection operation in America. This

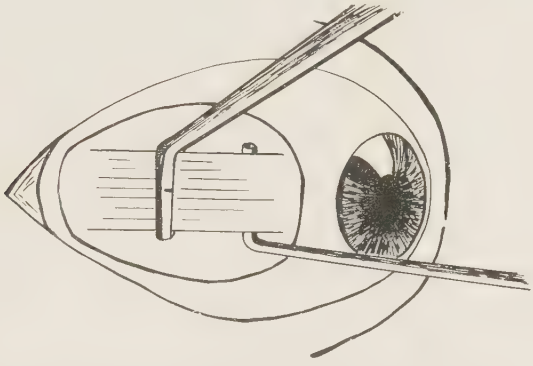

Fig. 87.—Reese's resection. Passage of the strabismus hook under the entire muscle. (From De Schweinitz, *Diseases of the Eye.*)

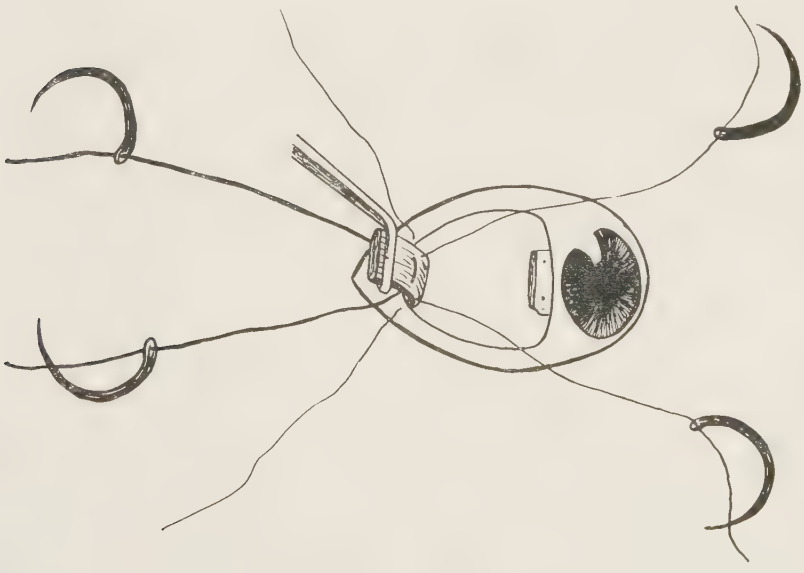

Fig. 88.—Reese's resection. Showing the three necessary sutures. (From De Schweinitz, *Diseases of the Eye.*)

was described by him in *New York Medical Journal,* January, 1912, and is given as follows by De Schweinitz:[68]

"*Reese's muscle-resection operation.* Dr. Robert G. Reese operates as follows, the description of the operation being in his own words:

" 'For the external and internal rectus make a vertical incision in the *conjunctiva* 6 mm. from the corneoscleral margin, commencing at the

level of the upper corneal border, and extending to the horizontal plane of the lower border.

" 'At the upper and lower limits of the incision just made grasp the tissue anterior to the sclera with forceps and open with scissors, directing their point away from the muscle. This procedure allows the passage of the strabismus hook under the entire muscle.

" 'When the muscle is held on the hook, dissect all the conjunctival and subconjunctival tissue back to the canthus, exposing the bare muscle completely.

" 'The lateral invaginations of Tenon's capsule, which are attached to the tendons of the ocular muscles, must be dissected free and clear.

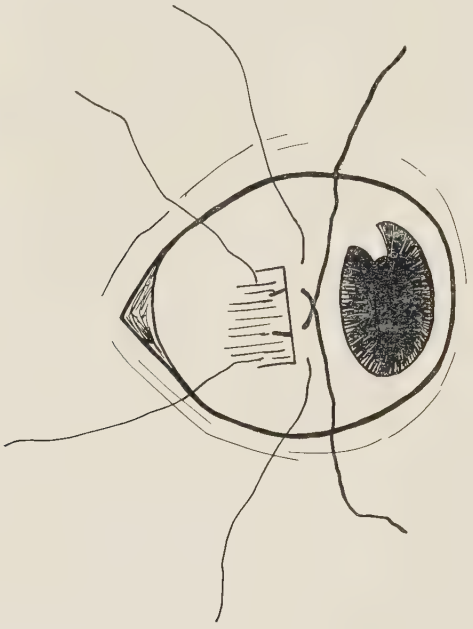

Fig. 89.—Reese's resection. Showing the muscle resected and sutures in scleral stump. (From De Schweinitz, *Diseases of the Eye.*)

" 'One blade of the resection forceps is then inserted beneath the muscle at a right angle to its course, so that the groove on the blade lies directly over the middle fibers of the muscle. Clamp the forceps to the last notch, and do not let its grasp include anything but muscle.

" 'Sever the muscle 2 mm. from its scleral attachment, leaving a stump, so that the resected end can be sewed to its original insertion. Free the belly of the muscle from any scleral adhesions. Three sutures are necessary.

" 'Put the sutures in, commencing with the middle, which is a No. 3 braided silk with a needle on each end. Pass one needle through the scleral surface of the muscle posterior to the blade of the forceps and 4 mm. back of the point of resection, and 1 mm. to the side of the groove on the forceps; then pass the other needle the same way, but to the other side of the groove making a loop with the suture on the scleral

surface of the muscle. As the needles pierce the muscle, let them include the dissected edge of the subconjunctival and conjunctival tissues.

" 'The two wing sutures are No. 5 silk with a single needle passed *first* through the upper and lower part of the dissected conjunctiva and episcleral tissues, including the superior and inferior border of the muscle, and slightly posterior to the loop made by the middle suture.

" 'Cut the muscle anterior to the sutures, leaving at least 2 mm. in front of the loop.

" 'Insert the two needles attached to the middle suture 2 mm. apart, through the center, and the two other needles through the upper and lower edges of the scleral stump. These needles should include the conjunctiva as they pass from behind forward.

" 'Tie the middle suture first in a loop, and do not use a surgeon's knot, as it will not pull up well. The lateral sutures are next tied. No supplementary conjunctival sutures are necessary.

" 'The middle suture is removed in ten days, and the others can be taken out any time after forty-eight hours, or if left in they soon fall out.

" 'The eye operated upon only is bandaged and is dressed daily for five days, when boric acid bathing three times a day is ordered; and if the eye is not overcorrected, the correcting lenses are ordered for constant use. On the other hand, if there is any undercorrection, a mydriatic is used and the proper glass is worn constantly.

" 'The operation consists in resecting the muscle only, and not cutting out any of the other tissues of the eye. It is a myectomy, because in no degree of squint however slight, will removing the tendinous portion of the muscle be sufficient.' "

As early as 1910 I became convinced that more definite squint surgery was essential; and earlier squint surgery was imperative. I had fallen into some of the pitfalls of tenotomy and abandoned it. I had used the various advancements and found them indefinite, difficult, or inaccurate. Worth's had its scleral stitch which was not always easy to introduce, neither did it always hold. Reese's resection was good, free from danger, but was difficult to measure, and the amount of correction it gave was limited. Verhoeff's advancement had its scleral stitch. However, it was effective, and lent itself to fairly accurate measurement.

It soon became evident that some means must be devised that would furnish a sure anchorage for the resected muscle, and at the same time entail the least possible danger. After trying various sutures, scleral and otherwise, it was found that the tendinous stump afforded the ideal anchorage. By leaving 1½ to 2 mm. of the tendinous stump, one is enabled to place a double-armed suture through its scleral surface (see Figs. 95 and 96) without fear of harm, which will have tensile strength enough to pull the eye into any position and hold it there without tearing out.

The next essential was to devise some means of measuring the actual amount of shortening done. This required an unusual exposure of the

muscle to be shortened, and a means of measuring this shortening. I devised a pair of calipers to measure the distance from the cut end to where the sutures were to be placed. This is now determined before the patient enters the operating room. This depends upon the degree of squint present. The degree of squint is measured frequently before the patient is sent to the operating room, usually during a period of six months. A printed slip is sent with the patient, and on this slip the degree of squint and the amount of shortening to be done are indicated. The calipers are set by the operating room nurse as per the above instructions. Here we have exact measurement of the degree of squint before operating and exact operative procedure, and have a right to expect definite end-results.

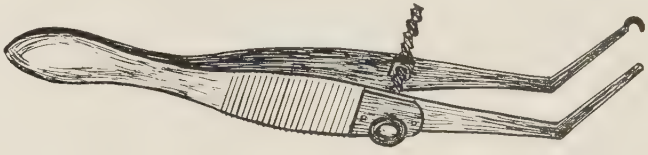

Fig. 90.—Calipers.

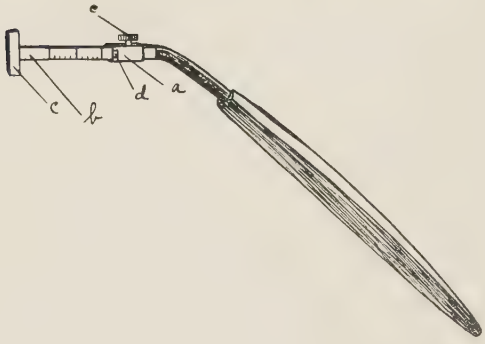

Fig. 91.—Wilkinson's strabismus rule for measuring accurately in mm. the shortening done in squint operations. *a*, sliding sleeve; *b*, shaft bearing millimeter scale; *c*, T-shaped piece at end; *d*, square pointer; *e*, set screw. (Service Instrument Co., Washington, D. C.)

I devised an operation in 1912 in which the scleral stump was used as an anchor. In this operation the central suture was identical with the suture described by Duverger and Metty.[69] In addition to the central suture, I used two lateral sutures which were introduced from the canthal side of the wound through the conjunctiva, the capsule, the upper and lower portions of the muscle just in front of the central suture, and then through the conjunctiva near the upper and lower borders of the cornea, thus closing the conjunctival wound. This is an excellent operation and I still use it on selected cases.

The only objection to this operative method was that there was always some *slight* doubt as to the *measurement,* and a faultlessly exact

method was to be desired. In 1915, after using the above-mentioned operation for some three years, I devised an operation (see below), which I have now used on more than 400 cases with much satisfaction and increasing confidence.

In this operation the muscle is exposed and the exact amount of shortening is measured with either calipers or rule (see Fig. 90). The sutures tied, a second measurement is made to determine just exactly how much the muscle has been shortened. In all other operations this can only be approximated.

Having found it difficult to measure accurately with calipers the exact point at which the muscle suture was to be placed, I devised a strabismus rule (see Fig. 91) which is much more convenient and at the same time more accurate. It consists of an instrument about the weight and size of a strabismus hook with a cross bar about 8 mm. in length at the end. Above this bar on the shaft, beginning at 5½ mm., is placed a millimeter scale up to 15 mm. At the upper end of the shaft is placed a sliding sleeve. On the right-hand side of this sleeve is a set screw and in front at its lower point, is placed a square pointer 3 mm. long. To use this instrument, the pointer is set at the desired distance from the inside of the T end. The muscle is detached and held by the muscle forceps and the suture is placed through the stump ready to be passed through the muscle at a certain distance. The assistant now introduces this measure externally to the exposed muscle down towards its belly, while the surgeon makes slight traction on the muscle forceps. The pointer is made to lie on the forceps over the cut end of the muscle. The surgeon now introduces his suture through the muscle from the ocular side, passing the thread through either angle of the T at the end of the instrument.

The third essential was to devise some means by which the eye can be controlled, or its movements so restricted, that the sutures will not give way or tear out. To restrict the ocular movement and relieve the stress from the stitches, a strabismus splint or brace was devised. (See cut and description.) I have now used this brace in more than 400 cases and in no case has any harm come from its use. It relieves the advancement sutures of all stress and stretches the overacting internus, and permits the advanced muscle to heal where it is left at the end of the operation, and permits the patient to be up and about, a great aid.

In my efforts to determine accurately the effect of a certain number of millimeters of shortening, I found some variance. In investigating the cause of this, I was convinced that where there was an unusually strong overacting internal rectus, due to anatomic overdevelopment, spastic conditions, or changes from deviation of squint, that the same degree of shortening would not produce the same amount of

correction as in a normal case. I then devised a pair of scales (see cut) to measure the pull or resistance of the internus. This I have used in the past 400 cases.

The scales are graded in grams. I have found that the average pull to a squinting eye is from 70 to 80 grams. Many will pull as much as 120 grams, and a few 150 grams (Fig. 92).

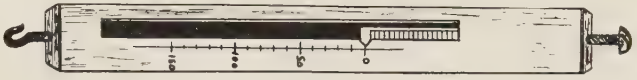

Fig. 92.—Scales for measuring pull of muscle.

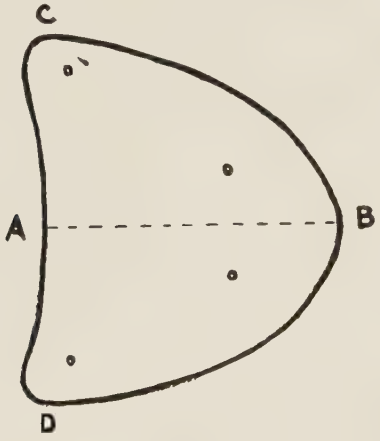

Fig. 93.—Strabismus brace; *A* to *B*, 11 mm.; *C* to *D*, 14 mm.; thickness, 0.5 mm.

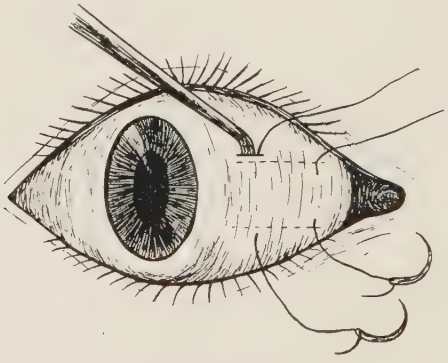

Fig. 94.—Method of introduction and position of stay sutures for strabismus brace.

The method of using the scales is as follows: The stay suture for holding the brace is placed underneath the tendon of the internal rectus muscle. (See Fig. 94.) This thread is knotted and the hook of the scales is caught in the thread. The eye is now pulled out until the edge of the cornea is brought to the outer canthus. The weight

of the pull required to bring the eye over is registered on the scales and recorded. In local anesthesia the patient is required to fix with the good eye as the squinting one is pulled outward until the cornea reaches the external canthus. Under ether the same procedure is followed; but, of course, we have no fixing eye. The degree of anesthesia probably causes a variation in the pull; however, I have not found it so. I feel certain that a markedly overacting muscle will be thus detected despite that possible error. In cases with slight overacting internal rectus I leave the brace in from ten to twelve days and take an extra millimeter of shortening. Cases which require 120 grams or more to turn the eye out will require a recession operation, or tendon-lengthening operation, such as Verhoeff's or Harman's.

In the event that there is overcorrection, which I have had in two cases, the method of correction is by leaving off the glasses and training the fusion faculty. In one case in which an error was made in setting the calipers by the inch scale instead of the metric, an overcorrection of 15 degrees was obtained. This was overcome by lengthening the muscle after the manner recommended by Bishop Harman.

Tropometric reading will usually indicate an overacting internus. In those cases in which there is found a deficient movement of the eye outward as shown by the tropometer, there is usually a greater pull found when using the scales.

The following description of my operation is taken from the *Journal of the American Medical Association,* 1922:

"Fig. 93 represents a strabismus brace which is to be anchored to the tendon of the internal rectus muscle. It consists of a semi-oval or shoe-heel shaped piece of flat lead, perforated in four places for the anchor sutures. Its dimensions are 14 mm. in its cross diameter, 11 mm. in the horizontal diameter, and 0.5 mm. in thickness. Its purpose is to stretch the overacting internal rectus and to fix the eye so as to prevent tearing of the sutures in the advanced external rectus muscle.

"Fig. 94 shows the method of introduction and the position of the stay sutures for the strabismus brace. A slight opening about 3 mm. long is made at either border of the tendinous insertion of the internal rectus muscle; a strabismus hook is inserted under the tendon, and the tendon is raised; then a thread, *A B*, is passed along beside the strabismus hook and out through the conjunctiva at the opposite edge of the tendon. Thus, the first thread, a No. 14 twisted silk, is placed underneath the tendon of the overacting internal rectus muscle; the second suture, a No. 8 twisted silk, is placed deep into the check ligament in the internal angle of the eye. These threads are now laid aside until after the resection of the external rectus has been completed, and then they are passed through the four perforations in the strabismus brace and tied.

"Fig. 95 represents the exposure of the external muscle, its clamping with muscle forceps, and the cutting of the muscle 1.5 mm. from its insertion.

"Fig. 96 presents the cut external rectus laid back; a double-armed 00 twenty-day chromic catgut is first introduced from within outward, one needle going through the upper fourth of the tendinous stump and the other needle through the lower fourth of the stump. The upper thread is now passed from within outward through the upper fourth

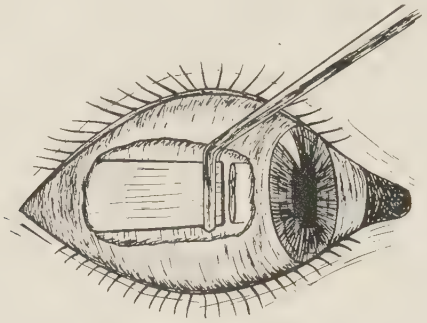

Fig. 95.—Exposure of external muscle, clamping with forceps, and cutting 1.5 mm. from insertion.

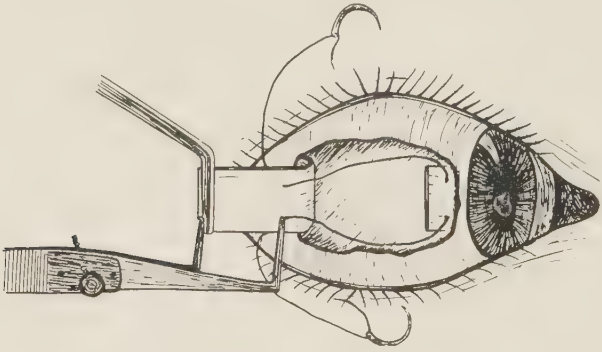

Fig. 96.—Cut external rectus laid back and main suture in place. Note calipers measuring the amount of shortening.

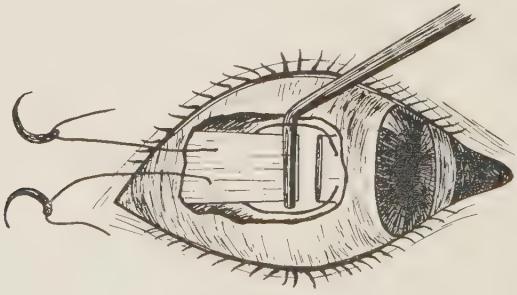

Fig. 97.—Thread in place and muscle held back in original position.

of the belly of the muscle at a distance measured by the caliper as herein shown, it having been previously determined how much shortening shall be done and the caliper set at that distance. The lower thread is now passed through the lower fourth of the muscle at the same distance from its cut end as the upper thread.

"Fig. 97 shows the thread in place and the muscle held back in the original position, in order to show better the exact method of placing this suture.

"Fig. 98 shows the eye pulled up straight, the end of the muscle brought up to or overlapping the corneal margin, and the suture tied. A firm hold is secured on the muscle, and yet there is no dangerous scleral stitch.

"Fig. 99 shows the muscle advanced, the main suture tied, and the placing of the two auxiliary modified Critchett sutures to guarantee no curling back of the cut ends of the muscle and also to guard against an accidental untying of the first or main suture. I use here a No. 5 silk suture. These two sutures pass through the conjunctiva and

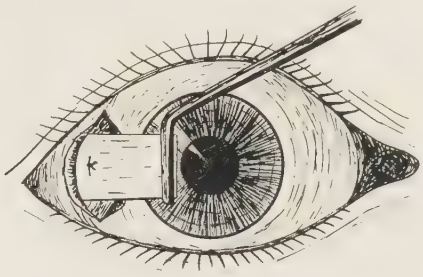

Fig. 98.—Eye pulled up straight; end of muscle brought up to or overlapping the corneal margin and the suture tied.

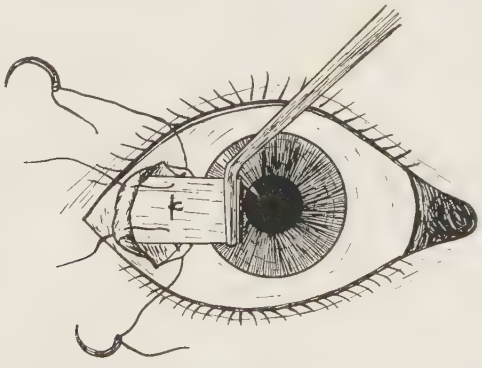

Fig. 99.—Muscle advanced, main suture tied, and two auxiliary modified Critchett sutures placed.

the capsule, through the upper and lower edge of the muscle respectively, in front of the main suture, and then through the conjunctiva. The main suture of 00 catgut is now cut, and the muscle is also cut about 3 mm. in advance of the Critchett sutures. The latter sutures are now tied, thus closing the conjunctiva.

"Fig. 100 shows the external wound closed, and the brace tied in the internal angle of the eye by the two anchor sutures.

"The eye is now closed and bandaged. Daily dressings of hot boric acid solution, atropin, and argyrol are used. The brace and the auxiliary or Critchett stitches are removed on the eighth day. The patient is permitted to be up and dressed after thirty-six hours, and may go about after the third day."

I have now used this method in over 400 cases, and can recommend it without hesitation to those who wish a definite mode of action to attain a definite end-result. This operation with the brace causes more reaction, but it is a much more definite procedure. Without the use of the brace I feel that it has advantages over other resections, in that the amount of shortening can be more accurately measured and it is mechanically stronger than any with which I am acquainted.

A Combination of Resection and Advancement.—There are a number of operations in which there is both a resection and an advancement. C. Worth's, Verhoeff's, and Jackson's operations are of this type. They are useful in cases in which a very large effect is to be secured from one operative procedure.

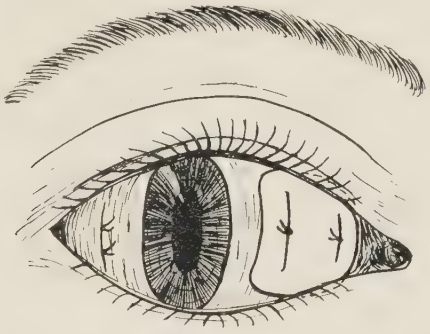

Fig. 100.—External wound closed, and brace tied in internal angle of eye by two anchor sutures.

Folding or Tucking Operations.—Of these there are two types. One, the shortening, is made by the use of a suture alone. In the other, the tendon is folded over an instrument and sutured in place. These operations are indicated in those cases in which there has been left only a slight degree of defect. The objections to these methods are, first, there is always a great doubt as to the amount of correction that will be secured. In the second place, the folding operation, when the thread alone is used, usually corrects only a very small amount of defect. When *tuckers* are used, more effect can be secured, but it is difficult to measure accurately the amount of shortening that has been done. In addition to this, there is usually an ugly *lump* caused by the tendinous fold, which remains for a long time. Much effect can be secured with these procedures in cases in which a tenotomy is performed on the antagonist, a procedure which should not be practiced as it also adds to our first indefinite result one more indefinite and also more harmful.

Of the tendon-folding operation with suture, that described by de Wecker[69a] was one of the first, and its modification is still being used.

A very simple single-suture folding operation is that which Lagleyze[70] described in 1891, which is as follows (Jackson):[71]

"The conjunctival incision is made concave to the corneal margin. In advance of the tendon insertion, a flap of conjunctiva covering the insertion is excised. The tendon is exposed and isolated on two strabismus hooks. A double-armed suture is introduced by passing both needles from the scleral surface outward through the tendon or muscle and conjunctiva sufficiently far back from the insertion;

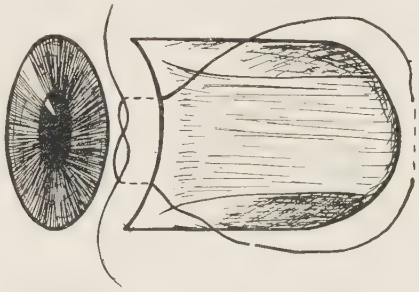

Fig. 101.—Folding of tendon as done by Lagleyze. (From *American Encyclopedia of Ophthalmology.*)

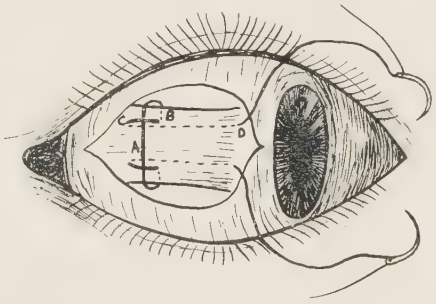

Fig. 102.—Stitch for folding tendon, proposed by Suffa. Both ends introduced. (From *American Encyclopedia of Ophthalmology.*)

one needle being passed 1 mm. from each margin of the tendon or muscle. The needles are then entered beneath the conjunctival flap adjoining the cornea, and superficially into the sclera, and brought out at the corneal margin, as shown in Fig. 101. The tightening of the suture folds the tendon forward almost to the corneal limbus. The suture is allowed to remain ten days to secure firm union."

Of this operation Savage[72] has the following to say:

"The best of all advancement operations, therefore the only one that should be done, is the 'flat advancement' without severing the tendon, devised by Lagleyze."

Certainly, one of the best tendon-folding suture operations is that described by Suffa[73] as follows:

"After exposing and isolating the tendon, a double-armed suture is laid across the tendon at *A*, Fig. 102, sufficiently far back from the insertion to give the required amount of shortening. The needle attached to the upper end of the suture is introduced beneath the tendon, which is made to pierce the scleral surface at *B*, at the junction of the upper and second quarters of the width of the tendon, in front of the suture *A*. It is then carried over the suture *C*, where the needle again pierces the tendon, from the conjunctival to the scleral surface, and is carried beneath the tendon to its insertion, where it pierces, emerging at *D*. The lower end of the suture is similarly

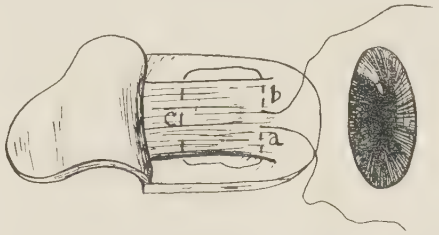

Fig. 103.—Catgut suture in position ready to be tied. When tied, the base, *c*, is approximated to the tendon insertion at *a* and *b*. (From Banister, *The American Journal of Physiological Optics.*)

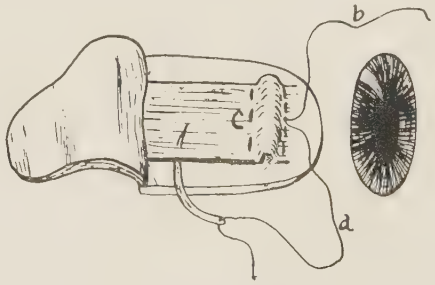

Fig. 104.—Suture tied. Needle reentered to make supplemental tuck. (From Banister, *The American Journal of Physiological Optics.*)

carried through at the junction of the lower and third quarters of the tendon, back, through, and forward to the insertion. The tendon is folded by tightening the sutures. The conjunctival opening is closed by a superficial suture on either side, being allowed to gape over the knot in the tendon suture."

The efficient catgut suture advancement operation which is presented by Banister is a modification of the operation as first introduced by Savage, and afterward by Valk. The accompanying illustrations (Figs. 103, 104, 105, 106) are self-explanatory.

O'Connor[74] described a unique operation for shortening the muscle, in which he split the tendon into four strands and formed around each

a loop of large catgut. This operation is rather tedious and none too accurate.

F. O. Schwartz,[74a] of St. Louis, has devised a very ingenious tendon splitting advancement operation, in which the tendon, after being exposed, is split down its center for about 1 cm. One-half is drawn up and the other down by means of threads, and mattress sutures are placed through gold plates to prevent tearing.

Briggs[75] described a very ingenious shortening operation by clamping a loop of the tendon with a small silver wire ring. The greatest objection to this operation is its inaccuracy as to exact amount of shortening done and the difficulty of removing the ring, as it at times becomes buried in inflammatory tissue.

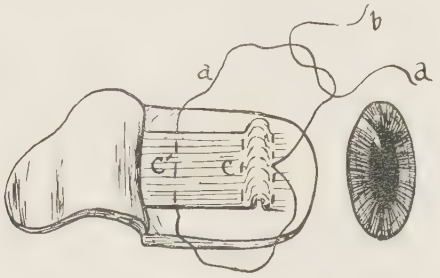

Fig. 105.—Supplemental suture in place, ready to be tied. (From Banister, *The American Journal of Physiological Optics.*)

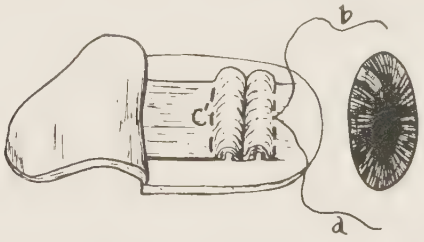

Fig. 106.—Supplemental suture passed and tuck completed. Note double tuck. (From Banister, *The American Journal of Physiological Optics.*)

Folding Tendons with Special Instruments.—Jackson[76] tells us that Maxwell[77] was one of the first to introduce an instrument for folding the tendon for the purpose of shortening the muscle in the relief of squint. This had two parallel fixed hooks and a central movable hook which slid between the two stationary ones. The tendon was picked up by the movable hook and drawn up between the two stationary hooks by means of a screw. The amount of shortening was indicated by a millimeter scale on the tendon folder. When a sufficient loop of tendon has been thus drawn between the two fixed hooks, a double-armed thread is passed through the double tendon from behind forward near the two fixed hooks. The sutures are tightened with a temporary knot, and

the hooks removed and the result *tested*. Additional sutures are placed if found necessary. Sutures are placed to flatten out the loop.

Clark[78] describes a very effective hook, which is similar to that introduced by Maxwell. This consists of a three-pronged hook controlled with a milled screw and ratchet. This instrument was improved upon by H. D. Bruns,[79] who also advised the use of a *guy suture* to bring forward and flatten out the tendon. His is one of the best folding or tucking operations.

Greene[80] in 1899 described a new type of folder. Todd[81] introduced a tendon folder. Bishop Harman[82] devised a very ingenious reefing forceps for folding the tendon. Bishop[83] recently introduced one of the best tucking instruments.

There are two objections to *all* forms of tucking operations on the tendons. In the first place, it is very difficult to tell *exactly* how *much* the muscle has been shortened. If the tendon is forced between the prongs of the forceps, much stretching must take place, and the tendon being folded, any error in measurement is always double. The advocates of this operation have maintained that there will be no torsion produced by this procedure. It would appear that this would occur as frequently, if not more frequently, than in a straight advancement, as any slight variation of the distance of the two lateral sutures from the cornea would be doubled, and hence would produce torsion. The second objection is that there is frequently quite a lump which remains for months. I have found that a fairly open advancement with definite measurement of the amount of shortening done has given far more constant effect, and without any greater reaction or danger. The claim made by advocates of folding operations that *no* harm can come from this procedure, that the worst that could happen would be a return to the previous status, whereas, if an advancement gives way, the second condition is worse than the first, does not obtain, if the sutures in the advancement are properly placed and protected by means of the brace or a recession as above indicated.

Operations Transferring Actions of the Muscle.—In paralytic cases efforts have been made from time to time to transfer the action of one muscle to the field of the action of the paralyzed muscle. Successes have been reported by Jackson, O'Connor, Peters, and others. The author has not had sufficient experience with these procedures to recommend them, but from a physiologic viewpoint they are worth considering. In paralytic cases, advancements of the antagonist muscle, with tenotomies in selected cases of the overacting muscle, would usually give satisfactory results. (See chapter on Paralytic Strabismus.)

Change of Insertion.—Jackson[84] recommends transplanting the tendon of a muscle to take up the effect of a paralysis of another. Stevens[85] and Jackson[86] recommend advancing tendons laterally to relieve dec-

linations which they consider potent etiologic factors in the production of squint.

Operations on Check Ligaments.—Motais, Kalb, and others have written several interesting articles upon the effect of cutting the check ligaments. Motais[87] has described in detail the operative procedure. Parinaud[88] advocates setting back the check ligament in certain conditions. The author feels that unless there is a marked restriction in the movements of the eyes, the same end can be more definitely obtained by other procedures which are not as complicated.

Capsular Advancement.—De Wecker,[89] in 1883, conceived the idea that if the capsule was advanced, leaving the muscle and tendon undisturbed, this would support and strengthen the action of the muscle. Knapp tried this, and, finding it inefficient, he modified de Wecker's operation, and converted it into a tendon-advancement operation, which was much more effective (Norris and Oliver).

L. W. Fox[90] has given us a very interesting description of his capsular advancement in his textbook on ophthalmology as follows:

"Tenotomy of both external recti muscles, making an opening through the conjunctiva, over the insertion of the tendon. Tenon's capsule is then stretched until the cornea is well into the inner canthus —this is an important part of the operation, and should be performed as follows: the strabismus hook, which is a large one, flat on its side, is inserted in the opened conjunctiva and Tenon's capsule, and with considerable traction all the tissues are stretched until the cornea is buried in the inner canthus. The stretching of the upper tissue has, as can be readily understood, a tendency to rotate the eyeball to a certain degree and leave the conjunctiva and Tenon's capsule intact below; to equalize the stretching the point of the hook is reversed, and the lower conjunctiva and capsule are also stretched.

"The upper suture is inserted through the conjunctiva and Tenon's capsule and across under the conjunctiva and Tenon's capsule, midway between the insertion of the superior rectus muscle and the margin of the cornea; a similar suture is passed through the lower margin of the conjunctiva and brought out midway between the insertion of the inferior rectus muscle and the margin of the cornea; this thread is then tied, and in like manner the upper thread; two more sutures are passed through the margin of the lips of the wound and tied."

There are such indefinite end-results obtained by capsular advancements when done alone that these procedures must be accompanied by tenotomy, and this, in turn, leaves an element of doubt. The trend should be toward adopting definite procedures to attain definite ends.

Recession Operations.—As to the indications for recession, or setting back of the muscle toward which it deviates, Beard[91] has the following to say:

"There is but one class of squint cases wherein the surgical treatment calls for an actual setting back of the muscle toward which the

eye deviates, viz., that in which the muscle is abnormally short. This is found in certain instances of congenital squint, and, exceptionally, in the case of middle-aged or elderly subjects with acquired squint in whom the defect has been of many years' duration. I refer particularly to those in which the length of the muscle is so inadequate that not only is the eye incapable of voluntary rotation in the opposite direction, but also refuses to so turn under fixation forceps, even with the subject in complete narcosis.

"To attempt advancement of the antagonist without a recession of this short muscle would result in the production of an unwarrantable degree of enophthalmos, and still greater limitation in the motility of the globe. Indeed, advancement of the opposite muscle is not needful save in a certain percentage of these cases, the recession alone being sufficient."

A. E. Prince, in the early eighties (Beard) published a paper in which he advocated a central suture in tenotomy. Colburn (Chicago)[92] in 1889 described an operation for controlling the tendinous insertion after tenotomy, calling it *curb tenotomy.* This is described as follows by Beard:[93]

"The tendon is exposed by a horizontal incision through conjunctiva and capsule, whose center is over the middle of the insertion. The tendon is caught upon a small flattish hook, which is shoved back to free the muscle from its sheath. A double-armed thread of No. 1 braided silk is put into the tendon 2 or 3 mm. behind the insertion by passing a needle down through close to each border, and the loop thus formed pulled down snug. The needles are then carried beneath Tenon's capsule, one above, the other below, just in line with the loop, given a good bite, and brought out through both capsule and conjunctiva. While the operator holds the thread, an aid rotates the eye far outward (it being the internus) in order to still further loosen the tendon from its surroundings; the globe is set free and the suture so tied as to leave a slight convergence. The same suture closed the opening in the membranes."

The profession is indebted to P. C. Jameson, of Brooklyn for introducing a definite procedure of recession. Dr. Jameson[94] published his operative procedure in the *Archives of Ophthalmology,* 1922, and his description of his operation is as follows:

"A conjunctival incision is made about seven millimeters long following the curve of the semilunar fold, the center corresponding to the caruncle. The ends of this incision are prolonged toward the cornea and in the direction of the fornici above and below. The flap is undermined to its base and turned over toward the cornea. The semilunar conjunctiva is also undermined in the direction of the caruncle, with care not to disturb the fascial trebeculae or areolar tissue on the surface of the muscle. (Fig. 107.)

"The muscle is now undermined, completely separated from the globe, and its capsular continuity severed above and below by incision carried back beyond the equator. A tenotomy hook is inserted to facilitate clean dissection, and Reese forceps, placed behind it,

grasp the muscle. The hook is withdrawn and the muscle severed from its insertion in the usual way.

"It is an advantage to have the operative field completely exposed and ample room to manipulate the needle. The sclera now exposed should be sponged and cleaned of blood particles and the outlying muscle fibers, if any, freely severed.

"The distance from the muscle insertion which the operator desires to recede the muscle end is now measured in millimeters and the point noted on the sclera. The remaining steps in the operation are very simple.

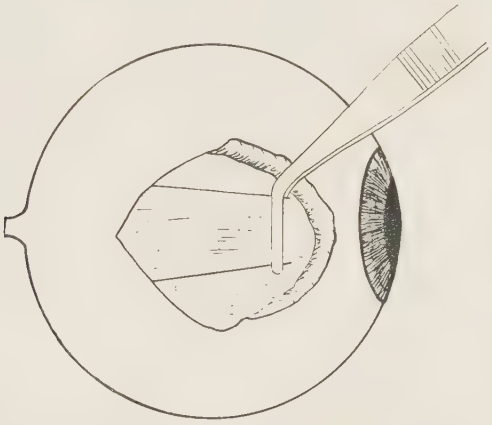

Fig. 107.—Muscle exposed. Capsular incisions pass backward beyond equator. (From Jameson, *Archives of Ophthalmology.*)

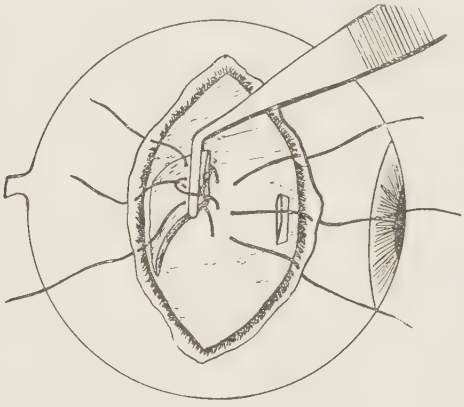

Fig. 108.—Muscle ready for recession. Three sutures introduced through conjunctiva, sclera, and again through conjunctiva. (From Jameson, *Archives of Ophthalmology.*)

"Three single-armed sutures are provided. The first, the central one, is introduced from without the conjunctival lip nearest the caruncle, then perforates the outer surface of the muscle end, just below the center behind the forceps. At this point to make for the utmost safety in the bite of the suture, the needle is put through the muscle from the under side above its first perforation (that is just above the

center of the muscle) and is brought out on the upper side of the muscle, the thread being looped around the thread of the first perforation. This simple suture is easily seen on the diagram. The sutures are now continued right on through the sclera and out and finally through the lip of the conjunctiva nearest the cornea. The two remaining sutures, about three millimeters above and below the center, are carried directly through the conjunctiva lip, muscle end, sclera, and conjunctival lip nearest the cornea. (Fig. 108.)

"The sutures are now carefully separated, the blood débris cleared away, the muscle approximated to its new scleral attachment.

"The tying of the sutures firmly holds the muscle to its new scleral position and at the same time closes the conjunctival opening. The central suture resolves itself into a surgical knot and tightly holds a bundle of muscle fibers in firm apposition to the sclera. If the conjunctival wound gaps between the sutures, it is wise to supplement surface conjunctival sutures. * * *

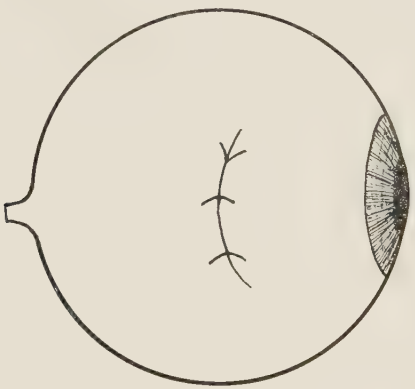

Fig. 109.—By tying the sutures the conjunctival wound is closed and the muscle, at the same time, is brought to its new point of attachment. (From Jameson, *Archives of Ophthalmology.*)

"Certain precautions should be observed in order to make a smooth and facile operation. The conjunctiva should be carefully separated, leaving the supramuscular tissues intact. The areolar tissue in which the muscle is deeply imbedded in the region of the caruncle should not be sliced. If this is done the muscle is endangered, the anatomy disarranged, and the reaction will be greater. (Fig. 109.)

"It is of great advantage to leave the mass of fat and fascia in which the muscle is embedded posteriorly intact. It not only preserves the anatomic relationship of the parts but leaves a compact mass of tissue in continuity with the capsular incisions above and below which unites and adds to the strength of the scleral attachment.

"The needles should be small, 10 millimeters from tip to eye, full curved and sharp. A large blunt needle would endanger the operation.

"We have found the ideal suture material is 00 or 000 10-day chromicized catgut. This has almost the strength of No. 1 silk. It

has the great advantage of nonirritative absorption in due time, so that digging for buried sutures is avoided. It holds equally as well."

Dr. Jameson states that before doing this operation the operator should master the technic of scleral suturing. This can be done on the enucleated eye.

There are two features of Dr. Jameson's operation that are of particular value. In certain cases in which there is a very definite overacting internus it becomes necessary to overcome in some way this overaction. This has been done heretofore by tenotomies, partial tenotomies, or muscle extension operations, all of which left much to be guessed as to what the ultimate effect might be. With a definite amount of recession, one can soon arrive at more or less accurate amount of correction to be secured. In other words, it gives us a *definite, measured* procedure to take the place of haphazard ones. In the second place, it secures all the benefit of a tenotomy without its

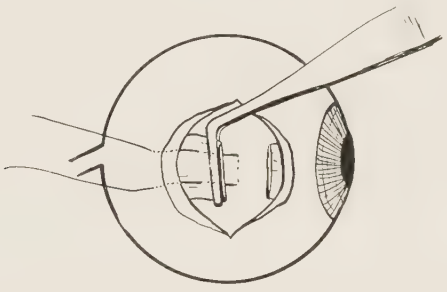

Fig. 110.—Wilkinson's modification of Jameson's central scleral stitch.

detriments, less restriction of convergence, less danger of divergence, and exophthalmos. In fact, with a measured recession and measured resection or advancement, it would seem that there shall be no excuse for permitting any one to remain cross-eyed at any age.

The modification (Fig. 110) of Dr. Jameson's excellent operation will obviate the scarring of the eye which is produced by his central stitch being placed over the end of the muscle; besides, it lends to strength as well as accuracy of measurement. I prefer the use of No. 1 braided silk for this central scleral stitch. This is brought out through the capsule and conjunctiva and should be removed on the eighth or tenth day. The two lateral sutures are placed as described by Dr. Jameson, and are tied first. The central portion of the conjunctiva is closed by a separate suture when deemed necessary.

Definite Operative Procedures.—It is deemed advisable to discuss definite operative procedures as applied to individual cases.

In slight degrees of internal deviation, say up to 18 degrees, in young subjects, an advancement of the external rectus of the deviat-

ing eye is the operation of choice. Exceptions to this rule will include eyes which are small, deep-set, and with small palpebral fissures. In such a case, and particularly in a case of a girl, in whom the matter of appearance should be considered, it would probably be best to divide the operative procedure between the two muscles, i.e., do an advancement of moderate degree of each externus. In a young child, say three to six years of age, with 18 degrees of squint which had not changed after six months' treatment with glasses, orthoptic exercise, etc. (see chapter on Nonoperative Treatment), I would advise doing an advancement of the external rectus of 10 mm., using the brace to prevent any giving way of the sutures, provided the tropometer showed normal excursions, and the eyes were sufficiently large and prominent. If the eyes were retracted and there was present some limitation of the external rotation, I would do a 3 mm. recession with not too wide lateral incisions, and a 6 mm. advancement.

In a case of, say 14 degrees of deviation, an advancement of 8 mm. accurately done, provided lateral rotations are normal, will give a satisfactory result. In low degrees of deviations, it requires relatively more advancement to correct a given defect than it does when advancing the muscle 10 to 12 mm. In other words, all other things being equal, it requires about 6 to 7 mm. of shortening to secure 10 to 11 degrees of correction, whereas, an advancement of 12 mm. will certainly correct as much as 20 degrees of deviation, when there is normal rotation. In the small, deep-set eye it is difficult to shorten a muscle 12 mm. and it is not desirable to do so, as too much shortening might cause some restriction in motion, as well as enophthalmos.

In patients with deviations of 20 to 30 degrees, it is usually advisable to advance both external recti muscles in prominent eyes, or do a recession and advancement operation on the same eye at the same time. It has been my experience that a recession operation with very slight or no lateral incision will correct 3½ to 4 degrees of deviation per millimeter of recession. If wide lateral cuts are made as suggested by Dr. Jameson, one obtains 4½ to 5 degrees of correction per millimeter of recession.

J. M. Wheeler, of New York, in a personal conversation, expressed the view that the recession of the internal rectus of more than 4 mm. might cause defective convergence. I think this view should be considered, and I would hesitate to recede an internal rectus muscle more than this amount in a case that has binocular vision, as convergence must be respected, it being one of the most important acts in the daily use of the eyes. Take, for instance, a case of 25 degrees deviation. Under ether anesthesia I would advance both external recti 8 mm., and place in the brace to be worn for a week (see description of brace, Fig. 93, p. 198). If the patient has 30 degrees of

squint, rotations normal, 9 mm. of shortening of each externus will be necessary. It is absolutely necessary to secure accurate measurements to obtain accurate results. Haphazard methods can but be disappointing. In case of amblyopia of the deviating eye, where there is serious objection to operating on the good eye, satisfactory results can be obtained by doing a recession and advancement. A case with 25 degrees of deviation would require 4 mm. of recession without lateral incisions, and 7 mm. of advancement. If 30 degrees of deviation is present, 4 mm. of recession with lateral incisions, and 8½ mm. of advancement, will be required.

For the correction of 30 to 40 degrees of deviation, the same rules hold good. The younger the subject, the more is resection indicated. Thirty-five degrees of deviation can be corrected with a double advancement of 10 mm. of each external rectus. Forty degrees of deviation can be corrected by a double advancement of 12 mm. each. In higher degrees of deviation, it is best to advance both externi at one sitting in the very young subject, and at a later date, if it is found necessary, do a recession of one of the internal recti, the amount to be determined by the degree of deviation left after three to six months. Glasses and orthoptic exercises, of course, must be used during this interval.

In older subjects with high degree of deviation associated with amblyopia, where there is serious objection to any operation on the good eye, it is permissible to recess the internal rectus 5 mm., also to make wide lateral incisions, and at the same time do a large advancement. Five mm. of recession with lateral incisions, in conjunction with resection, will correct about 25 degrees of deviation. Therefore, if we have an old amblyopic squint of, say, 45 degrees, with no hope of securing binocular vision, it would be advisable to do a recession of 5 mm. with lateral incisions, and a resection of the external rectus of 10 mm.

The following table I feel can be depended upon:

Advancement-resection with brace:

6	mm.	resection	will	correct	9°	of	deviation
7	"	"	"	"	11°	"	"
8	"	"	"	"	13°	"	"
9	"	"	"	"	15°	"	"
10	"	"	"	"	17½°	"	"
12	"	"	"	"	20°	"	"

Jameson's recession:

Without lateral incisions	*With lateral incisions*
3 mm. will correct 11°	13½°
4 " " " 15°	18°
5 " " " 19°	23° to 25°

Allowance must be made in all the above figures for any overacting muscle, or any particular control of the antagonist. They represent the findings in a number of cases accurately measured before the

operation (see chapter on Measurement of Strabismus), accurate measurement of the actual amount of shortening or recession done, and the end-result as found several months after the operation.

In cases with excessive convergence, without deficient divergence (Duane), a recession operation alone in low degrees of deviation, in lieu of the tenotomy, is indicated, but it is my belief and experience that in cases in which there is a possibility of binocular vision, the advancement operation is the operation of choice. I have not for some years used the tucking operation, on account of the difficulty of accurately measuring the amount of shortening that is being done, and in addition, the tucking operation, when done alone, has not been so universally satisfactory as the results I have been getting with the resection and brace.

Divergent Strabismus.—The same rules hold good in the operative treatment of divergent strabismus as given for convergent strabismus, with this exception: it will be found more difficult to advance the internal rectus than it is the external rectus, hence, about 10 mm. of shortening is all that can conveniently be made. This means that by the author's operation, only about 35 degrees of correction can be secured by the advancement of both interni when done alone. As these cases usually occur in grown subjects, it is advisable to do a tenotomy of the external rectus. Wootten[23] says:

"The surgical treatment of divergent strabismus is more complicated than that of the convergent variety and should depend entirely upon the character of the muscular anomaly that is causative.

"In regard to operative measures, when the squint is associated with myopia and insufficiency of converging power, advancement of both interni is the proper procedure, and is invariably followed by good results. When the deviation is associated with hypermetropia and an excess of diverging power, a free tenotomy of the externi repeated if necessary, will be equally successful. In the first class of cases, tenotomies of the externi, and in the second class, advancement of the interni will always result in failure."

It has been my experience that in the main Wootten's deductions are correct, but not more than 25 to 28 degrees can usually be secured by tenotomy of both external recti, and even this is not usually maintained, and an advancement must be done later to secure a satisfactory result.

Given a case of divergent squint of 15 degrees' deviation with pp at 3 to 4 mm., a liberal tenotomy with lateral cutting of the capsule will probably give a satisfactory result. If the deviation is 20 to 25 degrees, it will require a double tenotomy to secure the proper correction. A better procedure would be to do a liberal tenotomy of the externus, with a shortening of 7 to 8 mm. of the internus.

In a patient with 30 degrees of deviation who refuses to have the good eye operated upon, it is still possible by doing a tenotomy and a full resection up to 10 mm. of shortening to secure a good result. Where the eye deviates outward more than 30 degrees, and where the result must be secured by operating on only one eye, it is best to perform complete tenotomy of the externus, after stretching as recommended by L. W. Fox, with some other operation than that devised by the writer. The advancement of Verhoeff, or that of Jackson, or Worth should be used. By bringing the end of the muscle nearer the cornea, one can secure a greater amount of correction. These operations require a scleral stitch, which is more difficult to place, and is also more apt to give way, but inasmuch as the antagonist is freely tenotomized, the pull on the stitches is minimized, and particularly so if the eye is kept snugly bandaged for a few days. By means of this combination of operations, one can correct a very high degree of divergence by operating only on the deviating eye. Where a sufficient correction has not been secured, a second advancement can be done later, or a tenotomy and an advancement, if found necessary, of the straight eye might be considered. There is usually more redness and after-disfigurement in the way of thickened tissue following an advancement of the internus than that of the externus; however, this disappears in time under the influence of astringents.

Hypertropia.—The correction of a vertical deviation can be done at the time of the correction of the lateral deviation, or it may be undertaken later. A tenotomy of the superior rectus muscle, when overacting, is permissible, where there is a defect of more than 8 degrees of deviation. A tenotomy of the inferior rectus is *never justifiable.*

With a vertical deviation of 10 to 15 degrees, the underacting muscle should be shortened from 7 to 9 mm. just as recommended in the lateral deviations. I have very frequently operated on both the external rectus and the superior or inferior rectus, as the case demanded, at the same time, and have never had to regret this procedure. It saves much nervous anxiety for the patient, and I have seen no contraindications. The ratio of correction per mm. of shortening of the superior and inferior rectus muscles is practically the same as given for the externus (see table). If there be any difference, the effect is *slightly less.*

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CHAPTER XII

ILLUSTRATIVE CASES

In this chapter will be presented a few cases which will illustrate some points mentioned in the chapters on Nonoperative Treatment and The Operative Treatment of Strabismus. This is done to point out the defects of certain procedures and to indicate the best operative methods in certain definite cases. The essential facts only will be given in each case, in order to avoid unnecessary details which would detract from the report.

Case 1.—Bernard K. Age nine years.

First seen Oct. 4, 1923.

History: Eyes have turned in since the age of four years, following pneumonia; no history of squint in family.

Type of squint: Alternating.

Refraction: (Atropin)

R. $+3.0 \bigcirc +3.25$ L. $+3.0 \bigcirc +3.25$

R $+3.0 \bigcirc +.25$ cy ax $90°$ V = 6/6

L $+3.0 \bigcirc +.25$ cy ax $90°$ V = 6/6

Full correction ordered.

Tropometric reading: Normal.

Ophthalmoscopic findings: Negative.

Degree of deviation: 45° measured on four occasions.

Operation: June 23, 1925, each external rectus muscle was shortened 12½ mm.

Muscle pull with scales at operation: R 80 gm., L 100 gm.

Fusion: Grade 1 only.

Result: Dec. 5, 1925, the patient had 5° of exophoria without glasses; 8° with glasses.

Remarks: This child's parents were not interested in his fusion training. If he had been operated on two or three years sooner in all probability binocular vision would have been secured. It is to be noted that this child had a higher degree of squint under atropin. This is an indication for early operation. (Fig. 111.)

Case 2.—John A. Age six years.

First seen May 23, 1924.

History: Eyes have crossed since four years of age, following a blow on the head. No history of squint in family.

Type of squint: Alternating.

Refraction: R. $\frac{+2.50}{+2.50}$ L. $\frac{+2.50}{+2.50}$

R +2.50 V = 6/6
L +2.50 V = 6/6

Fig. 111.—Case 1. Before and after treatment.

Fig. 112.—Case 2. Before and after treatment.

Degree of deviation: May 23, 1924, R 40°, L 40°.
May 31, 1924, R 40°, L 40°.
June 14, 1924, R 42°, L 42°.
July 4, 1924, R 44°, L 44°.
July 9, 1924, R 44°, L 44°.

Tropometric reading: Normal.
Ophthalmoscopic findings: Negative.
Operation: July 9, 1924, each external rectus muscle was shortened 11½ mm.
Muscle pull with scales at operation: R 100 gm., L 110 gm.
Result: Nov. 3, 1925, the eyes are parallel without glasses and there is binocular vision with glasses. He has no enophthalmus.
Remarks: This case required several sittings for fusion training and bar reading to secure a satisfactory result. If he had been operated upon one year sooner the same result would have been easier to attain. (Fig. 112.)

Case 3.—C. R. E. Age four years.
First seen Jan. 3, 1926.
History: Eyes have crossed since the age of one year.
Type of squint: Alternating.

Retinoscopy: (After one week of atropinization)

$$R.\ \frac{-1.25}{-1.25} \qquad L.\ \frac{-1.50}{-1.50}$$

Full correction ordered.

Degree of deviation: Jan. 5, 1926, 38° in each eye; 40° under atropin.
May 1, 1926, 40° in each eye.
May 21, 1926, 42° in each eye.

Operation: May 21, 1926, each external rectus muscle was shortened 10 mm.
Muscle pull with scales at operation: R 90 gm., L 80 gm.
Result: Nov. 5, 1926, there were about 5° undercorrection without glasses, but with glasses there was binocular vision.
Remarks: This child had 40 to 42 degrees of deviation and each muscle should have been shortened 11½ mm. instead of 10 mm. as was done.

Case 4.—J. A. Age seven years.
First seen Aug. 25, 1925.
History: Eyes have crossed for two and one-half years previously following an attack of grippe. No history of squint in family.
Type of squint: Alternating.

Refraction: (After one week of atropinization)

$$R.\ \frac{+2.0}{+2.0} \qquad L.\ \frac{+2.50}{+2.75}$$

R +2.50 V = 6/6
L +2.75 ⌒ +.25 cy ax 90° V = 6/6
Full correction ordered.

Tropometric reading: Normal.
Degree of deviation: Aug. 25, 1925, 39°in each eye.
Aug. 31, 1925, 39° in each eye.
June 4, 1926, 40° R; 39° L.
Operation: June 7, 1926, each external rectus muscle was shortened 10 mm.

Fig. 113.—Case 5. Before and after treatment.

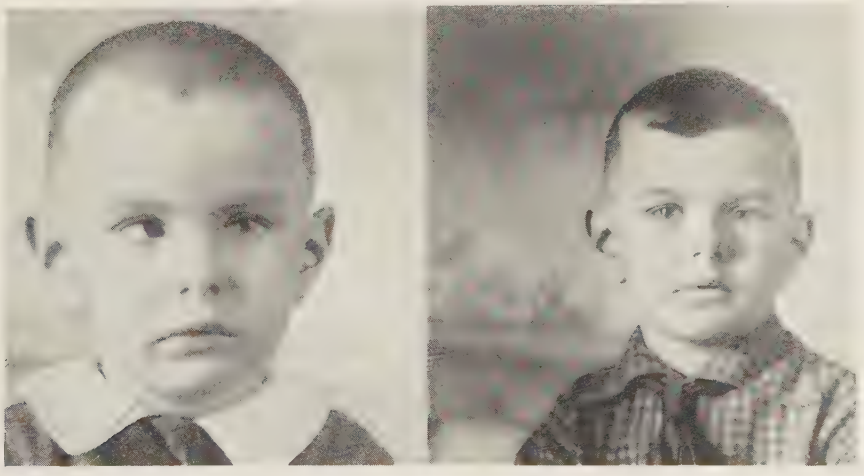

Fig. 114.—Case 6. Before and after treatment.

Muscle pull with scales at operation: R 70 gm., L 90 gm.
Result: Eyes are parallel; there is binocular vision with or without glasses.
Remarks: There are from 3 to 5 degrees of esophoria without glasses. The slight undercorrection would have been relieved by doing 11 mm. of shortening instead of 10 mm.

Case 5.—Katherine F. Age six years.

First seen Oct. 4, 1923.

History: Eyes have been crossed since birth. No history of squint in family.

Examination of fundus: No pathology; albinotic type.

Refraction: R. $\frac{+3.75}{+5.75}$ L. $\frac{+4.0}{+5.25}$

Degree of deviation: Oct. 4, 1923, 28° in each eye.
Oct. 20, 1923, 25° with glasses; 30° without.
Nov. 9, 1923, 29° without glasses.

Operation: Nov. 15, 1923, each external rectus muscle was shortened 9½ mm.

Muscle pull with scales at operation: R 75 gm., L 75 gm.

Result: July 22, 1925, there is binocular vision with or without glasses.

Remarks: This case has normal ocular movements and is satisfactory in every way. (Fig. 113.)

Case 6.—George L. N. Age seven years.

First seen Sept. 3, 1926.

History: Eyes have crossed since the age of one year.

Type of squint: Alternating.

Refraction: R. $\frac{+2.75}{+3.25}$ L. $\frac{+2.75}{+2.75}$

R +2.75 ⌒ +.50 cy ax 90° V = 6/9
L +2.75 V = 6/9
Full correction ordered.

Degree of deviation: Sept. 3, 1926, either eye 30° inward.
Sept. 10, 1926, either eye 29° under atropin.
Sept. 29, 1926, either eye 27° with glasses; 29° without glasses.

Operation: Jan. 7, 1927, recession of the internal rectus of 4 mm. was done, and the external rectus was shortened 7½ mm.

Result: Feb. 7, 1927, the eyes are straight; and there is second grade binocular vision with or without glasses.

Remarks: This child's fusion is not well established and at times the right eye will deviate a slight amount either in or out. If he had been operated upon two or three years sooner he would have attained binocular vision much more readily. He is still having his fusion trained. (Fig. 114.)

Case 7.—L. L. Age nine years.

History: Left eye was noticed to cross at times when three and one-half years old. By the time she was four and one-half years old it was crossed most of the time. However, the right eye would turn at times.

Type of squint: Alternating.

Refraction: R. $\frac{+2.0}{+2.0}$ L. $\frac{+2.37}{+2.37}$

R +2.0 V = 6/6
L +2.50 V = 6/5

Tropometric reading: Normal.

Degree of deviation: R 30° at each of several measurements; L 30°, and also turned up 6°.

Operation: Oct. 10, 1910, the left external rectus was shortened 10½ mm.

Result: There is binocular vision with glasses. Hyperphoria is reduced to 2°.

Later deviation: Jan. 15, 1926, the eyes are still straight with glasses, but have a tendency to cross when excited and glasses are off.

Jan. 16, 1926, there is 24° of esophoria which she was able to overcome with glasses and retain binocular vision.

Operation: Jan. 17, 1926, the right external rectus was shortened 9½ mm.

Result: March 1, 1927, there are 6° of esophoria and 1° of right hyperphoria; and patient goes without glasses on the street, has binocular vision at all distances, and is perfectly comfortable.

Remarks: This case illustrates the wisdom of the teaching of Valk, when he advised operating on cases of strabismus in which the defect is corrected by glasses. This is also practiced by Kearney. Practically all of these cases would be better off if their excessive convergence were relieved by a resection.

Many writers consider that binocular vision is seldom secured in alternating squint, but this has not been my experience. If seen early, the alternating cases are rather more hopeful, as, the visual acuity and the retinal images being about the same, they fuse the more readily.

Case 8.—Harold S. Age five years.

First seen April 19, 1922.

History: Eyes have crossed since the age of two years. Heredity: father is cross-eyed.

Type of squint: Monolateral; right eye fixing.

Retinoscopy: R. +1.75 / +2.50 L. +2.25 / +3.0

R +2.0 ⌒ +.50 cy ax 105° V = 6/9
L +2.50 ⌒ +.50 cy ax 90° V = 6/12

Degree of deviation: April 19, 1922, 40° in R or L.
April 25, 1922, 43° R or L under atropin.
Nov. 25, 1923, 43° R or L.

Operation: Dec. 12, 1923, each external rectus muscle was shortened 12 mm.

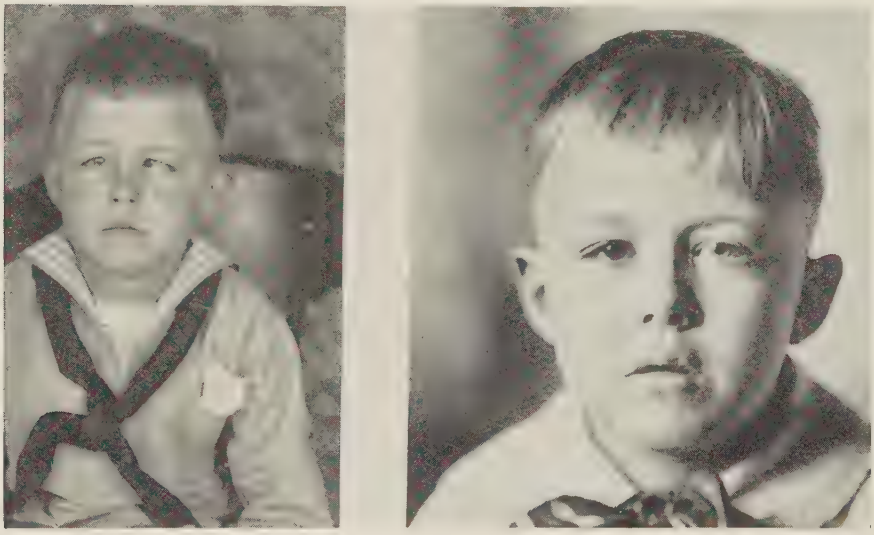

Fig. 115.—Case 8. Before and after treatment.

Muscle pull with scales at operation: R 80 gm., left 75 gm.

Result: Nov. 4, 1926, there are 5° of exophoria and ½° right hyperphoria. Binocular vision and acuity is now 6/6 in each eye.

Remarks: Here heredity did not prevent this child from securing binocular vision. (Fig. 115.)

Case 9.—Mundy S. Age seven years.

First seen Dec. 19, 1925.

History: Eyes have crossed since one year of age. No history of squint in family.

Type of squint: Monolateral; left eye fixing.

Retinoscopy: R. +2.25 / +2.75 L. +1.25 / +2.0

R +2.25 ⌒ +.50 cy ax 90° V = 6/60
L +1.25 ⌒ +.62 cy ax 90° V = 6/6

Degree of deviation: Dec. 19, 1925, 19° internal deviation R eye.
Mar. 27, 1926, 20° internal deviation R eye.
May 17, 1926, 19° internal deviation R eye.

Operation: May 17, 1926, this case was operated upon by Dr. Jameson of New York, who did his recession operation. (See Fig. 116.)

Result: There are 3 or 4 degrees of internal deviation without glasses.

Fig. 116.—Case 9. Before and after treatment.

Case 10.—Fred H. Age sixty-four years.

First seen Aug. 7, 1926.

History: Left eye has turned out since childhood. No history of squint in family.

Type of squint: Divergent, monolateral; left eye deviating.

Refraction: R +1.0 V = 6/9
L V reduced to counting fingers at three feet.

Degree of deviation: Aug. 7, 1926, L eye 30° out and 5° down.
Aug. 13, 1926, L eye 29° out and 5° down.

Operation: Aug. 14, 1926, recession of 5 mm. of the external rectus and 8 mm. of shortening of the internal rectus were done.

Result: Satisfactory; the eyes are practically parallel. (See Fig. 117.)

Remarks: In cases of this character a tenotomy of the external rectus would in all probability give as good results with much less traumatism.

Case 11.—L. B. M. Age thirty years.

First seen Oct. 8, 1926.

History: Eye began to turn out about ten years before and has steadily become worse.

Type of squint: Divergent, monolateral.

Refraction: R +1.0 ⌒ +.25 cy ax 90° V = 6/5
L +1.50 ⌣ +.25 cy ax 75° V = 6/6 + 1

Fig. 117.—Case 10. Before and after treatment.

Tropometric reading:*

	In	Up	Out	Down
R	50	35	60	50
L	40	35	60	50

Degree of deviation: L eye diverges 26° on looking at distance; pp 3½ inches.

Operation: Dec. 31, 1926, complete double external tenotomy was done.

Result: Feb. 27, 1927, pp is 1¾ inches; there are 6 degrees of exophoria for distance (Maddox rod); a 6 degree prism corrects lateral movements and a 2 degree prism corrects the vertical deviation. The patient is comfortable and satisfied.

*Stevens has given as normal for tropometric reading:

Up	Down	In	Out
33°	50° +	48 to 53°	48 to 53°

Remarks: If this case had had a tenotomy of the left external rectus with a resection of the internal rectus of the same eye at the same time, the ultimate results would have been more nearly perfect. This was explained to him before operating, but inasmuch as his duties would not permit of the extra loss of time required to do an advancement, he chose the tenotomies.

Case 12.—Julia B. Age five years.

First seen June 11, 1921.

History: Left eye turned in at times during the past year. It is now turned in most of the time.

Type of squint: Monolateral; left eye deviating.

Refraction: R. +3.50 / +4.50 L. +3.75 / +4.75

R +3.50 ⌒ +1.0 cy ax 95° V = 6/5
L +3.50 ⌒ +1.25 cy ax 85° V = 6/6
Full correction ordered.

Tropometric reading: Normal.

Ophthalmoscopic findings: Negative.

Degree of deviation: L eye 15° inward.

Fusion: Binocular vision (with glasses).

Result: Aug., 1922, there were 12° of esophoria with glasses.
Dec., 1926, there were 2° of esophoria with glasses.

Remarks: If this case had been neglected, she would in all probability have developed a permanent squint with the loss of vision in one eye, and it would have required an operation to relieve her deformity. I could report numbers of such cases.

Case 13.—Alice C. Age four and one-half years.

First seen June 10, 1921.

History: Right eye has turned during the past week. This came on suddenly.

Type of squint: Monolateral; right eye deviating.

Refraction: R. +2.25 / +4.0 L. +2.25 / +3.50

R +2.25 ⌒ +1.75 cy ax 90° V = 6/9
L +2.25 ⌒ +1.25 cy ax 90° V = 6/9
$+1.75_s$ combined with the above cylinders were ordered.

Tropometric reading: Excessive convergence in each eye.

Result: The deviation disappeared under a few treatments of fusion training, the use of glasses, and general tonics.

Remarks: The attached photograph shows the results obtained. This is what early care can do for these cases. (Fig. 118.)

Case 14.—M. B. Age twenty-nine years.

First seen April 23, 1923.

History: Suffers from frequent headaches and a drawing sensation in the eyes. Right eye has a tendency to turn when she is tired. She has noticed this for the past year, and the condition is getting worse.

Type of squint: Intermittent, divergent; right eye deviating.

Refraction: R +.25 ⌒ +.25 cy ax 45° V = 6/6
L +.25 ⌒ +.25 cy ax 90° V = 6/5

Degree of deviation: 26° of divergent squint; pp 2 inches on effort.

Fig. 118.—Case 13. Before and after treatment.

Operation: July 25, 1925, tenotomy of each external rectus was performed.

Result: July 30, 1925, there were 8° of esophoria for distance; Mar. 2, 1927, 3° exophoria for distance.

Tropometric reading: March 2, 1927, normal. Patient comfortable.

Remarks: The effect of tenotomy of one or both of the external recti is usually very satisfactory, where the deviation is not higher than 25 to 28 degrees, provided there is good converging power present. If there is a deficient convergence an advancement or resection is always indicated. In fact, the ultimate result is usually more satisfactory when a proper resection is done, but a tenotomy of the external is such a simple procedure and fraught with so little danger and discomfort that it will remain a favorite procedure.

Case 15.—B. E. Age fifty years.

First seen May, 1922.

History: Left eye has turned in since childhood following a fall down the stairs.

Type of squint: Internal, monolateral; left eye deviating.

Fixation: Good in each eye.

Degree of deviation: Left eye 38° in.

Refraction: R +1.75s V = 6/6
L +3.50s V = 6/12

Tropometric reading: Right eye, normal;

	Up	Down	In	Out
L	35	50	65	20

Muscle pull with scales at operation: L internus 128 grams.

Fig. 119.—Case 15. Before and after treatment.

Operation: Aug. 11, 1922, the left internus was exposed and lateral incisions made as recommended by B. Harman (see chapter on The Operative Treatment of Strabismus), until the pull was reduced to 65 grams. Each externus was then shortened 10 mm. and the brace used. The accompanying picture speaks for itself. (This operation was performed under ether.) (Fig. 119.)

Remarks: This case illustrates what can be done by means of accurate measurements, where the patient is under ether, notwithstanding the fact that Landolt, Wells, Meller, et al., contend that no one can secure definite results by any definite amount of shortening.

Case 16.—E. L. Age twenty-one years.

First seen May 21, 1921.

History: Right eye turned out since five years of age. The right eye is used only when the left eye is covered.

Type of squint: Divergent, monolateral; R eye deviating.

Fixation: Good in each eye.

Degree of deviation: July 12, 1921, R 35° out; L 38° out.
Aug. 4, 1921, R 40° out; L 39° out.
Aug. 9, 1921, R 40° out; L 40° out.

Operation: Aug. 16, 1921, each internal rectus was shortened 11 mm. (No tenotomy of external.)

Refraction: R. $\frac{-.75}{-2.75}$ L. $\frac{-5.50}{-5.50}$

Fig. 120.—Case 16. Before and after treatment.

(Sept. 12, 1921)

R $-.75 \bigcirc -1.75$ cy ax 80° V = 6/30
L $-4.50 \bigcirc -.50$ cy ax 150° V = 6/61

Result: March 2, 1927, there are 6° of exophoria and 2° of left hyperphoria.

Tropometric reading:

		In	Out	Up	Down
(Same date)	R	50	42	20	60
	L	53	48	20	58

Remarks: This case illustrates what can be accomplished by resection of the interni without tenotomy of the externi. Observe how nearly normal the tropometric reading is. (Fig. 120.)

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